The Canadian Medical Association Journal

JUNE, 1953 • VOL. 68, NO. 6

THYROID DISEASE IN NEW ZEALAND*

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I HAVE CHOSEN for the subject of this lecture thyroid disease in New Zealand. In 1920 after an absence of five years I was impressed by the widespread evidence of thyroid enlargement in the streets of my home town of Christchurch. Not the least of the many advantages of travel is this opening of one's eyes to the obvious and habitual in a familiar environment. With the co-operation of many colleagues in fields as diverse as geology, chemistry, physics and many departments of that conglomerate of sciences that we call medicine, a long term research was embarked on. The immediate objectives were to determine the incidence of thyroid disease in New Zealand and its distribution; to study the etiology of goitre and the physiology and pathology of the thyroid gland. In the thirty years of continuous work since the study was commenced some progress has been made. As in all research however, more problems have arisen than have been solved. Fascinating by-paths have unexpectedly opened up and the temptation to follow some of them has been hard to resist. I propose to trace the main direction of the work and to mention some of the by-paths.

THE SITUATION OF NEW ZEALAND

May I be forgiven if I remind you of the exact location of my country. It is not part of the great Australian continent. The Dominion of New Zealand lies some 1,200 miles South East of Australia, separated by one of the most turbulent portions of the Pacific Ocean named after the great Dutch explorer, Tasman, the first European to sight our shores in 1642. This Pacific Ocean which washes your shores as well as ours is the biggest thing on our planet. It is twice the size of the Atlantic. The Dominion of New Zealand consists of two chief islands lying almost antipodal to Spain stretching between latitudes 34 and 47° South and comprising an area of 100,000 square miles. Nowhere within these islands can one be more than 100 miles from the sea. Constantly are we New Zealanders reminded of the truth of Seneca's comment, "Beyond all things is the ocean".

Many scientists have expressed surprise that endemic goitre should be prevalent in such a maritime environment. This belief is apparently based on the fallacy that sea air itself supplies adequate iodine for the needs of the thyroid gland. In fact, sea air at the most can supply not more than 3 to 4 micrograms daily, an amount too small to be of much significance in an iodine-low environment. Sea water itself has a comparatively low iodine content, not more than 0.02 milligrams of iodine per litre. It is not until the plant life of the sea concentrates the iodine that it becomes available in physiological quantities. The avidity of marine plant life for iodine is of the same magnitude as that of the thyroid gland for the iodine of the blood. The cycle of iodine in the life of the sea is of great interest. The phytoplankton, rich in iodine, sinks to the floor of the ocean and serves as the food for the invertebrates of the bottom fauna which in turn supply iodine to the bottom feeding fishes. Fishes vary markedly in their iodine content according to their dietary. Only the bottom feeders or those feeding on seaweeds have a high iodine content in their flesh. Fishes therefore are not necessarily a rich source of this element. Seaweed is a much richer and more dependable source and though seaweed itself varies notably in its iodine content, any species dried can be relied upon to be at least a thousand times richer in iodine than any other food.

Unhappily the average New Zealander unlike the ancient Greek does not eat seaweed and his consumption of iodine-rich fishes is limited by both availability and cost.

*The Banting Memorial Lecture delivered at Toronto, April 29, 1952.
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Canad. M. A. J.
June 1953, vol. 68

THE NEW ZEALAND ENVIRONMENT

The geological history of New Zealand is a long and complex one. Though it contains in its fabric rocks of great antiquity, considered as a geographical unit the country is relatively young. It is not an oceanic island but the eastern edge of an ancient continent long since disappeared. After many vicissitudes its final emergence from the ocean and its isolation from all other land masses occurred in the cretaceous period. The pleistocene glaciers before their final retreat left an indelible mark on many New Zealand soils and thus in the fullness of time on our subject of thyroid disease in New Zealand. The Tuatara (*Sphenedon Punctatus*) the only living member of a long extinct order of reptiles bears witness to the final separation of the country from all other land masses in the golden age of reptiles before the emergence of the mammalia.

Lying wholly within the temperate zone and in an area of prevailing westerly winds, the climate is a cool, temperate, moist one without marked seasonal variation in either temperature or precipitation. The mountainous nature of the country which is one of its most striking characteristics affects the distribution of the rainfall to a considerable extent but secures abundant sunshine.

THE COMING OF MAN TO NEW ZEALAND

About the tenth century of our era, man first came to New Zealand. Despite the Kon Tiki epic the evidence is conclusive that our Maori people came initially from Tahiti and not from South America.

These master mariners, the Vikings of the Sunrise, as my old friend and colleague Sir Peter Buck designated them, sailing by sun and moon and stars, learned in all the language of the sky and the ocean and using signposts unknown to us, movements of the clouds and of the birds, the colours and flora of the ocean currents, traversed this vast ocean mass with amazing precision. Over a period of some four centuries a series of migrations of these Polynesian people came to New Zealand, the final and largest of which left Tahiti about the middle of the fourteenth century. Although they left no written records they handed on their traditions most faithfully by word of mouth. This verbal tradition records that goitre was already in evidence among certain of the tribes before the European era. Whatever may be the reliability of this tradition the thyroid gland of the modern Maori living in an endemic area responds in the same manner as does the European, as seen in Fig. 1 which depicts a large goitre in a pure blooded Maori woman living in an endemic area in the North Island of New Zealand.

THE COMING OF EUROPEANS TO NEW ZEALAND

Great Britain proclaimed sovereignty over New Zealand on January 30, 1840 when organized settlement by Europeans of British stock commenced. The first record of the appearance of goitre among the colonists appears to be in 1882 when Dr. Nedwill, the medical officer of

Fig. 1

health of Canterbury in his annual report stated: "Goitre has been of frequent occurrence in and around Christchurch and it will be interesting to watch as the district becomes better drained if this complaint will become less common". The next reference is in 1888 when Dr. Hacon records that goitre was widespread in the population of the whole of the province of Canterbury. In 1910 Professor Colquhoun, professor of medicine in the Medical School circularized the medical profession of New Zealand enquiring as to the incidence of goitre. From replies embracing over 200 towns and districts he concluded that goitre was now widespread throughout the whole country. In some districts it was exceptional to find a young woman without goitre.

In 1920 Dr. Eleanor Baker of the School Medical Service and myself examined 15,000 school children in the age group 5 to 12 years in Canterbury and Westland to determine the incidence of goitre. 32% were found to have markedly enlarged glands and a further 29% showed glands sufficiently palpable and visible on deglutition to constitute pathological enlargement. This survey was extended throughout New Zealand and over 90,000 children were examined. The results confirmed the findings of Colquhoun that endemic goitre was widely prevalent in the country as a whole.

TABLE I.

MORTALITY FROM DISEASES OF THE THYROID

Period	*Male*	*Female*	*Total*	*Rate per 10,000 mean population*
1889 - 1893	3	13	16	0.05
1894 - 1898	14	36	50	0.14
1899 - 1903	12	60	72	0.18
1904 - 1908	14	95	109	0.24
1909 - 1913	17	147	164	0.32
1914 - 1918	23	187	210	0.39
1919 - 1923	43	198	241	0.40
1924 - 1928	56	271	327	0.48
1929 - 1933	46	260	306	0.43
1934 - 1938	75	340	415	0.56
1939 - 1943	94	370	464	0.60
1944 - 1948	53	298	351	0.43
	450	2,275	2,725	

The influence of puberty was well seen by the examination of secondary school children in the age group 12 to 18 years, the incidence of goitre in girls rising to 80%. The increased demand for thyroid hormone was clearly seen in athletic boys, their glands being markedly larger than in the physically less active group.

In 1927 I found 68% of 121 young women of 18 to 22 years of age employed in a factory in Christchurch to have large goitres. At the same time at a maternity hospital 60% of the 200 mothers admitted over a period of twelve months had well marked goitres and 8% of the babies born had enlarged thyroids. In a large mental hospital in Christchurch 58% of the women and 52% of the men had large and well defined thyroid enlargement. Examination of recruits at the time of Great War I again revealed the prevalence of goitre. 1,500 men out of 130,000 examined were rejected for active service on account of goitre. There could be no doubt that the endemic was still present and was widespread.

In no part of the country has the severity of the endemic been such as to lead to endemic cretinism.

THE INCIDENCE OF HYPERTHYROIDISM

We found that an accurate assessment of the degree of hyperthyroidism in a community was much the more difficult task. The clinical recognition of the disease itself is fraught with difficulty and problems of nomenclature are substantial. The vital statistics for thyroid disease in New Zealand for both mortality and morbidity as revealed in the admissions to public hospitals are available since 1889. While these do not give a complete picture they do reveal the trend of thyroid disease in this country. In Table I the mortality is shown in five yearly age groups by sex with the rate per 10,000 of population. (This Table presumably refers mainly to toxic goitre.)

The progressively rising rate is evident until the quinquennium 1944-1948 when a decline has apparently set in. The sex differential is clearly evident. Table II shows the morbidity

TABLE II.

MORBIDITY FROM DISEASES OF THE THYROID

(*Admission to public hospitals*)

Period	*Exophthalmic goitre*			*Rate per 10,000 mean population*	*Total diseases of thyroid*			*Rate per 10,000 mean population*
	Male	*Female*	*Total*		*Male*	*Female*	*Total*	
1889 - 1893	No data			No data	—	—	64	0.21
1894 - 1898	"	"		" "	—	—	152	0.43
1899 - 1903	"	"		" "	—	—	175	0.44
1904 - 1908	"	"		" "	—	—	206	0.45
1909 - 1913	"	"		" "	No data			No data
1914 - 1918	43	223	266	0.48	154	804	958	1.74
1919 - 1923	87	375	462	0.76	269	1,234	1,503	2.53
1924 - 1928	212	1,180	1,392	2.07	455	2,347	2,802	4.16
1929 - 1933	360	1,939	2,299	3.20	668	3,395	4,063	5.65
1934 - 1938	448	2,195	2,643	3.54	866	4,280	5,146	6.89
1939 - 1943	710	2,531	3,241	4.20	1,262	4,314	5,576	7.23
1944 - 1948	676	2,798	3,474	4.29	1,031	4,269	5,300	6.55

Canad. M. A. J.
June 1953, vol. 68

for all diseases of the thyroid admitted to public hospitals, sub-divided into exophthalmic goitre and other diseases of the thyroid.

The most significant feature in this Table is the steadily rising incidence of the exophthalmic goitre admissions with as yet no evidence of decline. It is interesting to note in passing that surgical treatment of thyrotoxicosis commenced as early as 1896 when Closs, a Dunedin surgeon, records a successful thyroidectomy for Graves' disease. The surgical treatment of simple goitre was commenced in Dunedin as early as 1887 when Maunsell reports the surgical removal of a large simple goitre.

TABLE III.

MORTALITY

Period	*Male*	*Female*	*Total*	*Rate per 10,000 mean population*
1914 - 1918	No data			No data
1919 - 1923	1	18	19	0.03
1924 - 1928	11	15	26	0.04
1929 - 1933	20	31	51	0.07
1934 - 1938	20	39	59	0.08
1939 - 1943	18	30	48	0.06
1944 - 1948	16	45	61	0.08

TABLE IV.

MORBIDITY

			(*Admissions to public hospitals*)	
Period	*Male*	*Female*	*Total*	*Rate per 10,000 mean population*
1914 - 1918	0	5	5	0.01
1919 - 1923	1	12	13	0.02
1924 - 1928	18	23	41	0.06
1929 - 1933	26	24	51	0.07
1934 - 1938	28	42	70	0.09
1939 - 1943	29	48	77	0.10
1944 - 1948	34	57	91	0.11

MALIGNANT DISEASE OF THE THYROID GLAND

Drennan (1924) while holding the chair of pathology in New Zealand comments on the rarity of malignant disease of the thyroid despite the high incidence of endemic goitre. The situation has not changed throughout the years as will be seen by Table III which shows the mortality in five yearly groups by sex from 1914 to 1948 and from Table IV which shows the morbidity as shown by public hospital admissions.

The incidence in lower animals.—The lower animals are not exempt from thyroid disease. I have found goitres sometimes of large size in sheep, cattle, pigs and dogs. Of particular interest has been the occurrence of epidemics of thyroid enlargement in lambs. Thyrotoxicosis in animals is rare but I have seen it in a fox terrier dog and occasionally in race-horses.

The Pathogenesis.—We first set out to test the validity of the centrury-old theory of Chatin's that endemic goitre was the result of iodine deficiency. It is remarkable for how many years this theory languished when one studies the admirable series of papers published by Chatin between 1850 and 1876 supporting this contention. Apparently it required the discovery of iodine in the thyroid gland itself by Baumann for the full significance of Chatin's work to be realized. A large number of samples of water, soil, foodstuffs and urine from various districts of New Zealand and from the goitre-free islands of Samoa were analyzed for their iodine content. When these data were correlated with the goitre incidence among school children a very striking inverse relationship appeared which was too consistent to be fortuitous. The estimation of the iodine content of foodstuffs was found to be difficult and unreliable and as we found that the total iodine excretion in the urine throughout a twenty-four hourly period reflects with accuracy the iodine intake, and as this analysis proved to be an accurate one we adopted this figure as being the simplest method of determining the availability of iodine in a given food environment. We found that goitre is practically absent in regions where the average daily iodine excretion in urine is over 100 micrograms. Table V summarizes data to support this contention.

TABLE V.

URINARY EXCRETION OF IODINE IN RELATION TO GOITRE

	Daily urinary excretion	
Country	*Goitrous region*	*Non-goitrous region*
New Zealand	25 - 57	
Samoa		146
Switzerland	17 - 70	
Ligurian Coast		112
Norway	28 - 87	173
Holland	29 - 51	186
Lettland	60 - 70	120 - 160
Combined data	17 - 87	112 - 186
Average	52	149

From these findings we concluded that in New Zealand iodine deficiency played a predominant part in the production of endemic goitre.

Other etiological factors.—The irregular and unpredictable epidemics of goitre in lambs referred to above had made us alert to the possibility that factors other than iodine lack were probably

operating. It was not until Chesney, Clawson and Webster in 1928 discovered that cabbage contained an active goitrogenic agent that our attention was directed to the presence of active goitrogenic agents in food. Early in 1929 we initiated a series of long-term experiments to see whether New Zealand cabbage contained goitrogenic power. Young rabbits were sent to various endemic districts to be fed predominantly on cabbage, and extensive tests were commenced in our own laboratory. In 1933 Hercus and Aitken reported that only slight activity could be demonstrated in New Zealand cabbage. In this same

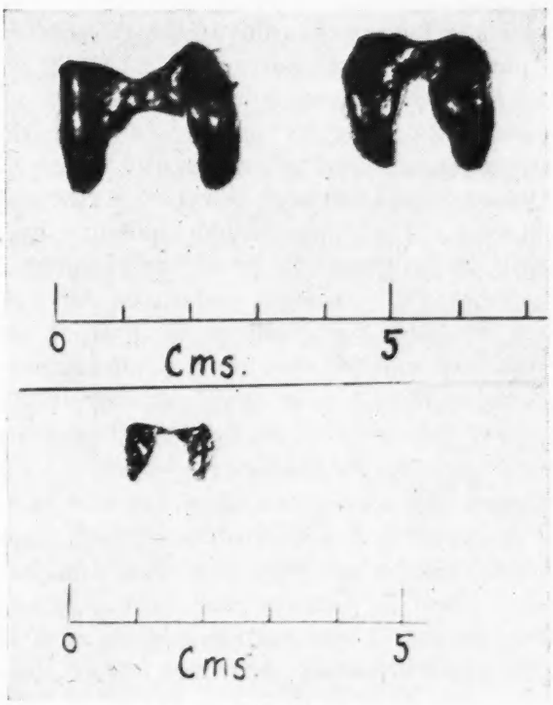

Fig. 2

year however an extensive outbreak of goitre occurred in lambs on an Otago farm where the ewes had for some months been fed exclusively on turnip roots. Young rabbits sent from our laboratory to the farm to be fed on the same turnip roots supplemented with hay and rolled oats within 60 days showed thyroid glands greatly enlarged and bright red in colour. The average weight of these glands was 1 gram as compared to the controls whose average weight was 0.1 gram. Fig. 2 contrasts the naked eye appearance of the normal with the enlarged glands. Histologically the enlarged glands were hyperæmic with high epithelium and no colloid in the vesicles. This was the first demonstration of goitrogenic activity in the roots of a *brassica* species.

In 1936 Hercus and Purves reported that goitrogenic activity was also present in rapeseed, cabbage seed and in the steamed seeds of both white and black mustard. At this stage in the research we secured the services of Mr. Kennedy to endeavour to isolate the goitrogenic factor. Kennedy proceeded on the assumption that the goitrogenic substance was a glycoside. He found that all the brassicæ seeds contained closely related glycosides which are broken down by enzymes in the seed to give mustard oils. Knowing that mustard oils react readily with ammonia to give thioureas and finding that the properties of the active agent in aqueous extracts of unsteamed seeds were consistent with thiourea structure, he proceeded to investigate the effects of both allylthiourea and thiourea on the thyroid glands of rats, and in 1942 he reported that their effect was to produce extreme hypertrophy and hyperplasia and almost complete loss of colloid in the affected glands. It was not however until 1949 that Greer, Ettinger and Astwood isolated the active agent 1-5-vinyl-thio-oxaladone from brassicæ seeds and later synthesized this substance in both its racemic and optically active forms, a brilliant piece of work.

By a curious coincidence at the same time as Kennedy independently discovered the goitrogenic action of thiourea, McKenzie, McKenzie and McCollum described the goitrogenic action of sulphaguanidine and Richter and Clisby discovered that phenylthiourea had the same properties. In 1941 Purves showed that the effects of these goitrogenic substances could be prevented by the administration of small doses of thyroxine and suggested that those agents acted by inhibiting the synthesis of thyroxine by the thyroid gland. In 1942 the clinical use of thiourea for the control of hyperthyroidism occurred to us as it was clear that these agents were acting as inhibitors of thyroid function. At this time in 1942 Dr. Crile, Jr. and Dr. Ernstine of the Cleveland Clinic then on service in New Zealand with the United States Navy visited the laboratory and in the light of our findings urged that clinical trials should be instituted. Unfortunately no thiourea was available and it was not until allylthiourea was synthesized in the chemistry department of this University that clinical trials commenced in October, 1943 shortly before the appearance of Astwood's masterly contributions on the use of thiouracil and thiourea for this purpose. Continued study on the use of the

Canad. M. A. J.
June 1953, vol. 68

thiourea drugs has been in progress in our thyroid clinic since this time. Since 1946 we have used methyl-thiouracil exclusively 200 mgm. daily in divided dosage until the thyrotoxicosis is controlled followed by a daily maintenance dose for at least six months of 50 to 100 mgm. In co-operative and properly selected patients the results have been most encouraging and in substantial agreement with those reported in the now extensive literature.

Meantime histological studies on the effects of rapeseed on other tissues were commenced. We were fortunate at this time to secure the services of Dr. W. E. Griesbach of Hamburg. He at first concentrated on the changes in the cytology of the pituitary and in 1941 he reported the marked increase in the number and size of the pituitary basophils identical with those first discovered by Rogowitsch after thyroidectomy in 1889. It was also found that hypophysectomized rats showed no thyroid reaction to the rapeseed and that the administration of iodine diminished but did not prevent the development of thyroid enlargement. Kennedy and Purves (1941) also recorded that the seeds produced histological changes in the adrenal cortex and delay in the development of the ovaries of immature females.

Later Griesbach and Purves found that these histological changes could be correlated with an increased secretion of thyrotrophic hormone and that these basophil cells responded to a small degree of thyroxine deficiency and were apparently concerned with thyrotrophic hormone secretion. They also reported that acidophil cell degranulation appeared in extreme thyroxine deficiency and was associated with failure of secretion of growth hormone.

In 1951 Purves and Griesbach continuing their investigation of the pituitary basophils found that they could be clearly differentiated into two functional groups by means of the McManus periodic acid Schiff reaction for glycoprotein. In one group the intensity of the glycoprotein reaction followed closely the gonadotrophic hormone content of the gland while in the other group of cells the intensity of the glycoprotein reaction was correlated with the thyrotrophic hormone content. From the nature of the specific responses of the two types it was concluded that the one type was exclusively gonadotrophic in function, and the other exclusively thyrotrophic. They also suggested that the Gomori stain is a specific stain for the thyrotrophic hormone.

The production of thyroid tumours.—During the course of the rapeseed experiments Griesbach, Kennedy and Purves (1945) found that long-term administration resulted in the formation of thyroid adenomata and they concluded that the induction of these tumours was the result of long continued stimulation by excessive amounts of thyrotrophic hormone, not to any direct carcinogenic agent. In 1946 Purves and Griesbach found amongst a group of thirty rats in which thyroid tumours had been induced by administration of thiourea for a period of nearly two years, that two animals had developed malignant thyroid tumours as evidenced by their invasion of the thyroid veins and by the presence of numerous metastases in the lungs. It was later found that these tumours could be transplanted into thyroidectomized rats and that transplantation was only successful when the recipient animal was kept in a state of thyroxine deficiency. The transplanted tumours grew slowly but progressively, invading the surrounding tissue and producing metastases, but they failed to cause the death of the host. If thyroxine was injected the tumours all regressed suggesting that a high level of thyrotrophic hormone was essential for their development in conformity with the Halsted principle.

During the course of a long series of transplantations over a period of three years, three different histological types of tumour appeared. One of these variants assumed more malignant characteristics. It was transplantable in rats with no thyroxine deficiency. A striking feature of the tumour was its rapidity of growth and its lethal effect in many cases on the host. In one case the host was killed in as short a period as three weeks and at autopsy the animal showed an extreme state of emaciation with atrophic muscles, thin, soft bones and a tumour weighing 50 gm.

Thanks to the splendid co-operation of the staff at Harwell radio-active iodine became available to us early in 1951 and an investigation of the iodine metabolism of these tumours with radio-active iodine was commenced. The original transplanted tumours had an iodine metabolism quantitatively equal to that of the animal's own thyroid and tumour transplants into totally thyroidectomized animals produced enough thyroxine for the animal's needs. The malignant variant however showed no selective iodine concentration. The availability of radio-active iodine has enabled us to develop its clinical use

as a diagnostic test in patients suffering from thyroid disease and in the investigation and treatment of patients suffering from recurrent thyrotoxicosis and from thyroid neoplasms.

PROPHYLAXIS OF ENDEMIC GOITRE

Iodized salt was introduced into New Zealand officially on a voluntary basis in June 1924. While this marked a definite forward step the salt was inadequately iodized as only one part of potassium or sodium iodide was permitted in 250,000 parts of salt. Even when used for both table and culinary purposes this supplementation only provided a maximum of 30 micrograms of iodine daily. As most people used it only for table purposes the supplementation was probably not more than 6 micrograms. In 1927 extensive enquiries from grocers and from a representative sample of homes in Canterbury revealed that only 5% of the salt bought for domestic purposes was iodized. A vigorous educational campaign by the Department of Health led to its more extensive use and by 1934 it was estimated that 30% of the domestic salt sold in New Zealand was iodized.

Since 1922 we had been carrying out several large scale prophylactic experiments in a large mental hospital in Christchurch, in orphanages and in the nurses' home in Dunedin Hospital using salt iodized to the level of one part of potassium iodide in 20,000; the salt being used for both table and culinary purposes. This degree of supplementation we had found to be necessary to raise the average twenty-four hourly urinary excretion of iodine to 100 micrograms. The results were entirely satisfactory and comparable with those obtained by Marine and Kimball in the United States and by Klinger and his co-workers in Switzerland. The therapeutic results were also encouraging and in some of the patients in the mental hospital, remarkable.

Continuing pressure on the Department of Health led in 1940 to the standard of iodine being raised to one part of iodine or potassium iodide per 20,000 parts of salt. Although the use of this salt was probably not operative before 1942 a notable decline in the incidence of thyroid enlargement in school children is already evident. In the Annual Report of the New Zealand Department of Health for 1951 Dr. Tolley reports that in a survey of over 7,000 children in Canterbury no large goitres were seen and only 2% showed thyroid enlargement readily visible on inspection. It appeared that 80% of the families in the district were now using iodized salt at least for table purposes. The Thyroid Research Committee of the New Zealand Medical Research Council has recommended that this salt should become the standard domestic salt of New Zealand and that only those asking specifically for non-iodized salt should receive it, but this policy has not yet been adopted.

CONCLUSION

We conclude that Chatin's original hypothesis that endemic goitre is a deficiency disease is true for New Zealand and that its ultimate control is practicable if the deficient iodine is supplied. The causation of hyperthyroidism remains unsolved. Its incidence in New Zealand appears to be correlated to some extent with endemic goitre but this is by no means a clear cut finding. There is no history of epidemics of hyperthyroidism nor does the disease appear to vary in severity from one part of the country to another. As in diabetes, so in goitre: while much is now known of their etiology the unknown remains greater than the known. The exact mode of action of insulin and thyroxine and their inter-relationships with one another and the whole of the endocrine system is unknown.

Endless horizons open before us. There is only one well marked trail to further progress. There are no short cuts. The research worker whether in Canada or New Zealand must have something of the same vision, the same tenacity of purpose and the same courage as the man whose memory we honour on this occasion, the late Sir Frederick Banting.

Thirty-five healthy premature infants, varying in age and weight, were studied for liver function by the bromsulphthalein excretion test; there is a progressive maturation of liver function with age in prematures, although this does not always correlate with weight. Premature infants of 18 to 28 hours of age excrete bromsulphthalein in a pattern similar to that of adults with impaired hepatic function. The normal adult pattern is attained at 6 to 8 weeks of age, but the 45 minute retention values became normal at 3 weeks of age. This progressive development of normal excretion is due to a progressive maturation of the anatomic and physiologic capacity of the liver. The increasing size of the capillary network in the liver as occurs with advancing age increases the hepatic blood flow and hence more blood is exposed to the liver sinusoids for BSP removal. – Obrinsky, W., Denley, M. and Brauer, R. W.: *Pediatrics*, 9: 421, 1952.

Canad. M. A. J.
June 1953, vol. 68

HUMAN BOTULISM IN CANADA*

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PREVIOUS REPORTS from these laboratories[1, 2, 3] have emphasized the probability that unrecognized cases or outbreaks of botulism occur in Canada. This communication illustrates anew that human botulism is not as rare a disease in this country as the hitherto low recorded incidence suggests. Another fish-borne fatality in British Columbia is described—the third episode in this Province bacteriologically proved due to *Clostridium botulinum*, type E. In addition, three clinically typical but bacteriologically unproved outbreaks, two of which occurred in Saskatchewan, and one in the North-West Territories, are outlined.

A FATAL CASE OF TYPE E BOTULISM DUE TO PICKLED TROUT

The following episode occurred at Natal, British Columbia, a small community about 20 miles from Fernie, in the southeastern corner of the Province. On September 23, 1952, a healthy, 42 year-old man, weighing about 140 lb., opened a jar of home-pickled trout cutlets, and ate most of the contents. A few hours later he vomited, and the following day was nauseated and constipated. Over the next two days, nausea and constipation persisted, and there was increasing general malaise. When seen by a physician on the fourth day after consuming the fish, the patient was depressed and miserable, and complained of "sore" throat; but the temperature, pulse and blood pressure were all normal, and no unusual physical findings were noted. (In retrospect, the doctor recalls some ptosis being present.) Next day the patient was admitted to the local hospital for observation; but not until the following morning (the sixth day) were many of the classical signs and symptoms of botulism apparent—inability to swallow, diplopia, ptosis, extremely dry tongue, and a practically paralyzed bowel and bladder. He was able to walk about, but was very weak. At this juncture, his physician wrote to the author at Vancouver, describing the case, suggesting a diagnosis of botulism, and forwarding a companion jar of pickled trout for bacteriological examination.

The patient had to be fed at first by stomach tube, and later intravenously. A stool sample was solicited, but none could be procured. Laxatives and enemas were without avail until the tenth day of illness. Then, within a few minutes after a dose of carbachol, the patient staggered to the toilet, leaving no specimen. Two days later, he was unable to leave his bed, and on October 7 he was transferred to a Calgary hospital, where specialists diagnosed bulbar poliomyelitis, despite negative cerebrospinal fluid findings. During these last days, although his arms and legs were not completely paralyzed, the patient moved very little. He died on October 11, 18 days after consuming the trout. The autopsy findings were reported consistent with a diagnosis of botulism, although cultures of the duodenal contents, examined locally, yielded no growth of *Cl. botulinum*. Sections of the central nervous system showed no microscopic lesions suggestive of poliomyelitis or encephalitis.

The following information was secured about the source, storage and preparation of the fish fillets. The trout had been caught about two months previously in the nearby Elk River by the victim, who was fond of fishing. His custom was to clean such fish on the river bank, after which they were either cooked and eaten with little delay, or stored in the household refrigerator for a few days until there was enough to put up in jars. This particular fish was kept in the refrigerator until the day it was pickled, when it was removed to an outdoor root cellar. The man's wife did the pickling, following a method handed down by her mother-in-law. The fish was made into patties, dipped in eggs and cracker crumbs, fried, and then placed in glass jars with sliced raw onions between each pattie. A hot vinegar and salt mixture was poured over the fish, and the jars sealed without further heating. The woman herself never ate such fish, as she disliked it. She had previously had to dispose of jars pickled in this way, because they had gone bad; but there had been no such trouble with this particular batch of several jars. Her sister's family had eaten the contents of one jar without ill effects, and other jars of the fish had been consumed at various times beforehand. Indeed, a friend of her husband, who ate a little fish from the top of the implicated jar, remained

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unaffected. The lid of this jar had apparently not been tight; but unfortunately none of the contents remained for laboratory examination, as the wife washed out the jar when the man fell ill.

Laboratory findings.—The doctor forwarded to us the only jar left of the batch. This was a 1-pint sealer with screw-on top, whose lid and rubber ring fitted tightly, and had to be pried off. It was filled to the brim with fish, pale in colour and firm in consistency, which looked neither raw nor scorched. Portions of sliced onions were noted, but no egg batter. The general appearance and odour were quite appetizing, with no signs of fermentation. A few ounces of pickling fluid, present in the bottom of the jar, had a pH 5.1. Mice injected intraperitoneally with 0.3 c.c. amounts of this fluid, and with similar amounts of a saline infusion prepared from several grams of the fish, showed no untoward effects.

There was no aerobic growth when comparatively large inocula (2 to 4 gm.) of the pickled trout were incubated in a glucose-peptone-beef infusion (G.P.B.I.) medium to which ground meat had been added. However, when this medium (boiled and then cooled to around 90° C.) was similarly inoculated, and incubated anaerobically at 37° C., the presence of gas-forming, non-proteolytic, sporulating, Gram-positive bacilli became evident after 3 or 4 days. By streaking on anaerobically incubated human blood agar plates, a culture having the colonial and biochemical characteristics[3] of *Cl. botulinum*, type E was isolated.

The original culture supernates proved non-toxic for mice; but after undergoing several daily transfers in the G.P.B.I. medium, a culture which had been left for a few days at room temperature (23° C.), and which displayed good growth and vigorous gas production, was shown to contain about 1,000 mouse M.L.D. per ml., neutralizable by type E antitoxic (rabbit) sera.

The laboratory findings, together with the clinical and epidemiological data, leave little doubt that this recent fatality was due to type E botulism, acquired from home-pickled trout.

TWO OUTBREAKS OF BOTULISM IN SASKATCHEWAN

CASE 1

On May 3, 1941, a 55-year-old woman and her two daughters (aged 21 and 14 years) fell ill at their home near Maple Creek, Saskatchewan, with abdominal pain and vomiting. They noted a gradual development of sore throat, blurring of vision with diplopia, and difficulty in swallowing, breathing and voiding. When admitted to hospital 8 days later, they showed marked weakness, dilated pupils, impaired visual acuity, and coated tongues. Their temperatures and urinalyses were normal. "Poisoning of unknown origin" was diagnosed, and symptomatic treatment administered. They improved sufficiently to be discharged from hospital on May 17.

Six days later, on May 23, the woman's husband, aged 59, and their son aged 28, fell ill a few hours after an evening meal of potatoes and home-canned fish. The onset was marked by severe abdominal pains, nausea and vomiting, which merged with a more acute and rapid succession of the symptoms shown 2 to 3 weeks before by their womenfolk—sore throat, dysphagia, blurred vision and diplopia, difficulty in urinating, headache, and pain in the chest. When admitted to hospital on May 25, their temperatures, pulse and respiratory rates were normal. Urinalysis disclosed nothing unusual. They were very weak and listless, their tongues dry and coated, pupils dilated, and voices hoarse. Dyspnœa was obvious, and soon became severe. Symptomatic treatment proved ineffectual, and their condition worsened rapidly. The son died on May 26, and his father next day, about 72 and 97 hours respectively after the suspected meal.

Following these events, the mother and daughters were readmitted to hospital for further investigation. They still displayed extreme weakness and general malaise, impaired vision and dryness of throat. After a further two weeks in bed, they were finally discharged, though not yet fully recovered, over 5 weeks from the onset of illness.

No details are available regarding the foodstuff involved in this remarkable situation of two groups within one family stricken by consecutive outbreaks of clinically typical botulism; but it would seem a reasonable conjecture that the same batch of home-canned fish was responsible for each occurrence. A more thorough investigation of the women's illnesses might have led to condemnation of the suspected foodstuffs, and possibly to laboratory verification of its botulogenic nature. The subsequent deaths of the men could thus have been averted.

CASE 2

On May 7, 1949, near Masefield, Saskatchewan, a rancher's wife, aged 32, prepared supper for the farm hand (21), the hired girl (18), and her own daughter (12), prior to a Saturday night trip to town with her husband and two younger children. After supper, the farm hand and the hired girl went to their homes for the week-end.

The meal had consisted of home-canned beef, potatoes, turnips, and rhubarb sauce. The beef was from a batch which had been put up as follows by the housewife, four months previously. Meat from the round off a hind quarter, purchased from a neighbour, was cut into small pieces, and packed into 5 or 10 lb. tin pails. With the lids off, the pails and their contents were roasted for 2 or 3 hours at 350° C. in the gas stove oven. When the meat appeared cooked, the pails were removed from the oven, the lids put on, and a wax seal poured over them. Meat thus canned had been stored in the basement. It had not kept well; but for the evening meal in question, the contents of a freshly opened can were sampled by the housewife, who considered the meat good enough to serve.

About 24 hours after the meal, the hired girl and the 12-year old daughter developed general weakness, which increased so much overnight that the former was unable to return to work on Monday morning. By this time both girls had blurring of vision and ptosis, and the

younger one's voice was hoarse. The mother and the hired man, who had been unduly tired the night before, now complained of severe fatigue. Next day, May 10, while the daughter's weakness progressed, she noted diplopia and dysphagia. She was therefore taken to the family physician, who found her temperature, pulse and respiration normal, and sent her home with a diagnosis of influenza, which was then prevalent in the district. On the same day, the hired man went to another physician, who likewise diagnosed influenza. During the next few days, the condition of all four victims worsened, their weakness increased still further, and dysphagia became more marked; and eventually they were all admitted to hospital—the daughter and the farm hand on May 12, the hired girl on the 13th, and the mother on the 16th. Finally, on May 19, the Medical Health Officer was consulted, and botulism was tentatively diagnosed.

When examined on this date, the patients showed to varying degrees the distinctive features of botulism. The two girls were obviously very ill, while the mother was least affected, presumably because she tasted only a small portion of meat. The farm hand, who was very sturdy, weighing 180 lb., never appeared more than moderately ill. Their temperatures and pulse rates were normal. The girls displayed a slightly raised respiratory rate, with some dyspnœa. *Progressive weakness* was in each case the predominant complaint in the early stages; and as long as six weeks after the onset, the two girls could not sit up for more than half a minute without fainting. *Constipation* was complete for periods ranging from 12 to 28 days after admission to hospital, despite administration of enemata and pituitrin; but none in this group suffered abdominal pain or bloating. Kidney function appeared normal throughout. Only one of these cases—the mother—experienced nausea and vomiting, on the second day of illness. *Dysphagia* came on gradually, involving solid foods first, and later, liquids. Four days after the onset of symptoms, the girls could not swallow fluids. In all cases, palatal reflexes were lost. *Aphonia* was confined to the two girls, who at the height of their illness could only whisper: previously, and later when their voices returned, they sounded hoarse, as did the other patients. Their tongues were coated, buccal mucosæ dry, and breaths foul-smelling; while in their throats ropy mucus collected, provoking an intermittent cough.

Ptosis, with dimness and blurring of vision, was definite even in the less severe cases; but the girls could hardly open their eyes sufficiently to get a glimpse of their hospital room, and were unable to read comics for about 7 weeks. Pupillary reflexes, to both light and accommodation, and also corneal reflexes, were lost. *Dilatation of pupils* and *lateral nystagmus* were displayed by all. Peripheral reflexes were normal, or only slightly diminished. Voluntary muscle tone and movements were normal. Cerebrospinal fluids showed no abnormality, and there were no complaints of headache or evidence of meningismus. Sensory changes were not apparent. The patients remained mentally alert throughout their illness. All four patients recovered, without apparent sequelæ; but definite weakness and lassitude still remained three months after the onset.

Twelve days after the toxic meal, an investigation was launched. At the farm, it was found that the original can, the source of the suspected meat, had been incinerated. Nine other cans of beef were found in the basement, one being of the same batch as the destroyed can. The wax seal had been blown on most of the cans, and the contents of all except one were malodorous, and obviously contaminated. Two were overgrown with mould, and one was "crawling with maggots". A spoiled jar of pickled beets was also discovered. The farmer conceded that his wife had told him the canned meat was not keeping well and some of it would have to be thrown out; but the investigators felt the woman withheld information from them about the condition of the suspected can of beef.

This episode forcibly illustrates the nature and extent of the hazard to which many householders, particularly in rural areas of Canada, may yet be subjected through inadequate knowledge and skill applied to the preparation of home-canned foods.

A FATAL OUTBREAK DUE TO SEAL MEAT IN THE NORTH-WEST TERRITORIES

On July 21, 1945, Eskimos arriving at the Lake Harbour detachment of the Royal Canadian Mounted Police, in the Eastern Arctic subdivision of the North-West Territories, reported that 7 natives had died earlier in the month at Markham Bay. All the deaths had occurred in one camp, which numbered about two dozen men, women, and children. There had been no contact between the stricken camp and any other camp or individual since May 9. On July 8, five members of one family, including the father (age 41), mother (36), two daughters (12 and 7 years) and a son (9 years), fell ill and all of them died; the elder daughter within a few hours, the younger daughter and the son on the 9th, and

the parents on the 10th. A 15 year-old girl of another family, and her 8 year-old brother, also took sick on July 8. The girl died on the 12th, but the boy recovered. The remaining case was the head man, aged 56, who became ill on July 10, and died on the 13th.

There was general agreement among the unaffected members of the camp, which included 3 men, 4 women and 8 children, that the symptomatology was uniform. As reported by these natives, and transcribed in the police report, the main features were "numbing of the throat glands, without pain or swelling. The throats of the sick ones seemed to be dead, and they were unable to eat because they could not swallow, nor were they able to drink or talk. . . . Towards death, the throat apparently closed somewhat, and breathing was very difficult, coming in gasps". According to Rawson,[4] who has briefly alluded to this outbreak as probably due to botulism, the chief complaints were extreme weakness, accompanied by dizziness, blurred vision, and dysphagia.

Although many details are lacking, it seems definite that the victims, most of whom lived in one tent, had eaten nothing but seal meat from a carcass which had been kept on the ground inside the tent for an unknown number of days. During mid-summer, seals are usually shot from a boat, and stored unskinned in the family tents, chunks being sliced off as required. Visiting between tents is common, and in times of plenty there is much community feasting, so that meat from a single seal could affect more than one family. Conversely, more than one seal might be implicated in a single outbreak of botulism. Carcasses are sometimes heaped in a pile on the shore for 2 or 3 days prior to being skinned and eviscerated by each owner, the offal being thrown to the dogs; while in some camps, the seals are kept together in the head man's tent for distribution by him. There is thus plenty of opportunity for either endogenous (intestinal) or exogenous (soil) contamination to spread from seal to seal. Whichever custom prevails, no attempt is made to keep the seal meat on ice. In these circumstances, after a few days in mild or warm weather, *Cl. botulinum* spores could certainly germinate and manufacture lethal amounts of toxin. The Eskimo's preference for raw, or even slightly putrefied protein, may then be his undoing. In the Lake Harbour episode, the meat was at most parboiled.

Any doubt that sea mammals can be the vehicle for botulism under Arctic conditions should be dispelled by Meyer and Eddie's report[5] of several deaths from this disease at Point Hope, Alaska, on the Chutkchi Sea, roughly 200 air miles north of the Arctic Circle, in 1947 and 1948. No food specimens were examined until 1950, when in the same locality a family of 5 became ill after feasting on uncooked beluga (white whale) flippers. Paralysis of the throat and diplopia were prominent symptoms, but the attacks were relatively mild, and all the victims recovered. The delicacy was described as follows:

> "The flippers are removed from the carcass of the whale, rinsed in sea water, and then placed on the rocks of the shore to dry. The partially dried flippers are then cut into strips and deposited in a 5-gallon can containing seal oil. The uncovered container is stored for several months in the hut of the owner. During this 'curing' or 'aging' process, the material contaminated with the dirt from the rocks is under anaerobic conditions. Elaboration of toxin is facilitated by the warm temperature of the hut. The beluga flippers in oil are considered by the Eskimo a delectable article of food and are usually eaten uncooked on special occasions."

Meyer and Eddie demonstrated the presence of *Cl. botulinum* type E toxin in a small sample of flipper, but were unable to isolate the anaerobe therefrom. Later, in our own laboratory,[6] from a similar sample of beluga kindly sent by Dr. W. H. Gaub of the Arctic Health Research Centre, Anchorage, Alaska, a toxigenic strain of *Cl. botulinum* type E was isolated.

DISCUSSION OF KNOWN CANADIAN OUTBREAKS OF HUMAN BOTULISM

The four newly described occurrences of botulism in Canada, and six outbreaks previously reported in the literature, are tabulated below. These ten episodes occurred between 1919 and 1952, and involved 56 persons, of whom 32 died. The case fatality rate, 57%, is closely comparable to the 65% rate indicated by Meyer and Eddie's[7] totals of 477 outbreaks, 1,281 cases, and 829 deaths for the United States during the period 1899-1949. But this similarity in average rates should not obscure the fact (illustrated in the table) that the mortality rate in a given outbreak may range from nil to 100%.

The disparity between the recorded incidence of botulism in this country and in the United States, to which attention has been drawn before,[2] is now considerably reduced; but the Canadian figures are still less than one-half those

calculated on a comparative population basis from United States totals. Moreover, within Canada, botulism has occurred very erratically. Except for one outbreak in Zurich, Ontario, the disease has apparently been confined to Northern or Western Canada, the North-West Territories having had one outbreak, the Yukon 2, Saskatchewan 2, and British Columbia 4 outbreaks. Only the last 4 of these have been bacteriologically identified.

The irregular geographic distribution of botulism throughout the world derives mainly from the interaction of such factors as climatic conditions, dietetic habits, and the numbers and types of *Cl. botulinum* spores in the regional soil. But the more populated southerly latitudes of Canada resemble large areas of the United States in their prevailing temperature ranges, and in standards and methods of food preservation, so that on this continent the major determinant of the various incidence of botulism may be toxigenic spore distribution. This supposition is to some extent supported by the findings in the only known extensive survey of North American soils for *Cl. botulinum*.

TABLE I.

BOTULISM OUTBREAKS IN CANADA

Year	*Cases*	*Deaths*	*Place of occurrence*	*Foodstuff implicated*
1919	23	12	Dawson City, Yukon	Commercially canned beets
1933	3	1	Zurich, Ontario	Home-canned tomatoes
1940	5	3	Whitehorse, Yukon	Uncooked salmon eggs
1941	5	2	Maple Creek, Saskatchewan	Home-canned fish
1944	3	3	Nanaimo, British Columbia	Home-canned salmon
1945	8	7	Markham Bay, N.W. Territories	Uncooked seal meat
1948	2	2	Grand Forks, British Columbia	Home-canned asparagus
1949	4	0	Masefield, Saskatchewan	Home-canned beef
1949	2	1	Vancouver, British Columbia	Home-pickled herring
1952	1	1	Natal, British Columbia	Home-pickled trout

Over 30 years ago, after examining nearly 2,200 soil specimens from many parts of the United States, Meyer and Dubovsky[8] concluded that the soils of Pacific Coast and Rocky Mountain States were heavily infected with *Cl. botulinum* spores, particularly type A. Extending their survey to Western Canada,[9] they found a similar situation. From 39 samples of soil, vegetables and green moss, collected in the vicinity of Vancouver, 6 type A strains were isolated: 5 samples yielded toxigenic cultures which could not be typed. Again, from 29 samples of Rocky Mountain soil collected near Glacier, B.C., and above Lake Louise, 5 type A and 3 type B cultures were isolated; while types A and B were both obtained from one sample, and toxigenic but untyped cultures from 3 samples. This high incidence (38.5%) of positive samples could largely account for the fact that 4 out of 10 Canadian outbreaks of botulism have occurred in British Columbia. However, it would have to be assumed that type E strains (unrecognized in 1922) were among the 8 toxigenic, untyped cultures isolated in the above survey; for of the 4 known outbreaks in this Province, only one has been due to type A, the others being of type E origin. This assumption seems justifiable, since in 1944 Dolman and Kerr[1] isolated a type E strain from the soil of a chicken run near Nanaimo, B.C., at the scene of a type E outbreak. Incidentally, in 1948, a type A strain was isolated in similar circumstances from the soil of an asparagus bed near Grand Forks, B.C.[2]

Meyer and Dubovsky found the intensely cultivated and fertilized soils of the Atlantic States less infected than the virgin and mountain soils of the far West, with type B spores predominant; while in the Middle States, the Great Plains and Great Lakes regions, and the Mississippi Valley, both types were sparsely distributed. Although they received insufficient numbers of samples from corresponding sections of Canada to warrant comparisons, their finding of only one positive specimen (type A) among 19 samples from various parts of Quebec does at least suggest a lower incidence of soil infection in that Province than in British Columbia. On the other hand, type A cultures were isolated from one out of only two specimens sent from Prince Edward Island, and also from Nova Scotia; while of two specimens from Ottawa, Ont., one yielded a type A culture, and the other a type B. In fact, there is no evidence that the botulism hazard can be dismissed as insignificant in any part of Canada. A new and thorough soil survey is long overdue,

as previously emphasized,[1] and should include specimens from the far North. The techniques should pay due heed to the relatively low heat-stability of type E spores; for the high proportion of Canadian outbreaks—6 out of 10—in which fish or a marine mammal has been implicated, might reflect a heavy incidence of type E spores in Canadian soil and littoral waters, since type E. botulism has so far been conveyed almost exclusively by fish.[6]

Despite its highly characteristic syndrome, botulism is liable to be mistaken for conditions ranging from poliomyelitis and myasthenia gravis to influenza or some form of chemical poisoning. Even when a reasonably thorough clinical examination is made, the requisite epidemiological approach is often lacking, particularly among specialists. In the type E fish-borne fatality at Natal, B.C., for instance, the practitioner's tentative diagnosis of botulism was overruled in favour of bulbar poliomyelitis. Again, Meyer and Eddie[5] have reported a type B fatality in California due to Liederkranz cheese, in which the diagnosis of botulism was disputed by a neurologist, who attributed the symptoms to "thrombosis of the basal artery, affecting the neck, throat and upper respiratory system".

Admittedly the botulogenic potentialities of such foods as pickled fish and soft cheeses are not yet sufficiently realized. Indeed, many current texts still contend that acidic foodstuffs are seldom vehicles for botulism. Such errors require time for correction. Meanwhile, general practitioner and clinical specialist alike may well join with the bacteriologist, the health officer and sanitarian, the public health educator and dietician, in reminding the general public of the mortal dangers sometimes inherent in home-preserved foodstuffs which have not been sufficiently heated prior to consumption.

SUMMARY

1. A fatal case of botulism due to home-pickled trout is described. *Cl. botulinum* type E was isolated from a companion jar of fish.

2. This represents the third fish-borne type E botulism episode in British Columbia in the past 8 years. Of the total 6 persons affected, 5 have died.

3. Two hitherto unrecorded outbreaks in Saskatchewan are reported. At Maple Creek, in 1941, 5 persons developed clinically typical botulism, and 2 died, apparently from home-canned fish; and at Masefield, in 1949, home-canned beef was responsible for 4 non-fatal cases.

4. An outbreak of botulism occurred among Eskimos at Markham Bay, North-West Territories, in 1945. Of 8 natives who fell ill after consuming raw or slightly cooked seal meat, 7 died.

5. In all, over the period 1919 to 1952, there have been 10 known occurrences of botulism in Canada. These have involved 56 persons, of whom 31 died. Only 4 of these episodes, all in British Columbia, have been bacteriologically identified.

6. By comparison with the United States figures, and from available data on spore distribution in specimens of Canadian soil, it seems probable that greater diagnostic alertness would reveal a higher incidence of human botulism in Canada than is currently apparent.

The author is glad to acknowledge his indebtedness to the following persons, without whose help this paper could not have been compiled: to Dr. V. L. Matthews, Regional Medical Health Officer, Department of Public Health, Saskatchewan, for an account of the two Saskatchewan outbreaks; to Dr. A. F. W. Peart, Chief, Epidemiology Division, Department of National Health and Welfare, Ottawa, and to Dr. N. A. Rawson, formerly Medical Officer at Chesterfield Inlet, Eastern Arctic, for information bearing on the outbreak at Markham Bay, North-West Territories. Thanks are also due to Drs. L. R. Amundsen and R. R. M. Glasgow, of Michel, B.C., for clinical details of the recent fatality at Natal, B.C.; to Miss Helen L. Byrt, R.N., Public Health Nurse at Fernie, B.C., for epidemiological enquiries relating thereto; and to Miss Helen Chang, M.A., for assistance in the laboratory investigation of this case.

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There are one-storey intellects, two-storey intellects and three-storey intellects with skylights. All fact collectors who have no aim beyond their facts are one-storey intellects. Two-storey men compare, reason, generalize, using the labours of the fact collectors as well as their own. Three-storey men idealize, imagine, predict; their best illumination comes from above through the skylight.—Oliver Wendell Holmes.

Canad. M. A. J.
June 1953, vol. 68

A NEW APPROACH TO INFANT FEEDING

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A RECENT ARTICLE on infant feeding,[1] advises that soft foods should be "started at three months, *when the infant can digest them,* but before he needs them for his nutrition." Another authority[2] suggests that the additions to the diet be made as follows:

Cereal	3- 4 months
Egg yolk	4 months
Vegetables	6 months
Bananas	4- 6 months
Fruit	6- 7 months
Meat	10-12 months

These statements embolden me to place on record the result of six and one-half years of experience in a newer method of feeding infants. Shortly after my discharge from the armed forces, with the co-operation of my more enlightened mothers, I suggested the earlier administration of all solids. I evolved the following system of infant feeding.

THE FIRST 7 DAYS

Orange juice.—Since orange juice is mainly water, it is given pure, sweetened with corn syrup or sugar from a sterilized cup with a sterile spoon. One-half teaspoonful per day is given and increased by one-half teaspoonful until at least 3 ounces is taken daily. In my experience orange juice is taken with delight at this age.

Vitamin D.—If in the concentrate, it is given by adding one drop to the orange juice and increasing by one drop daily until the necessary amount is being taken. Otherwise the usual method of giving cod liver oil is followed.

Cereal.—Cereal or Pablum is started at 7 days, one dessertspoonful or one tablespoonful is given as a start. The quantity is increased rapidly as taken or desired and is followed by the breast or formula. It is given at 10 a.m. and 6 p.m. although if the mother's milk is limited or failing in quantity, it may be given prior to each breast feeding. In this case it very often increases the production of maternal milk through permitting the continuation of nursing. I have not observed any difficulty in getting the baby to partake heartily of cereal at this age.

Formula.—At this stage, milk formula, if necessary, is given in the proportions of 1.5 milk to 1 of water with a total daily addition of 1 ounce of sugar.

12 TO 14 DAYS

Fruit.—As soon as it is seen that the cereal is well taken and to allow time for idiosyncrasies or allergies to manifest themselves, fruit is added to the cereal or Pablum. This may consist of crushed bananas, peaches, apple sauce, mashed prunes, pears etc. and it too is given at the 10 a.m. and 6 p.m. feeding, whether the infant is on cereal at each feeding or only at the morning and evening meal.

Formula.—At this time the formula is changed to 2 milk to 1 water. (I have strengthened the formula to these proportions at 10 days but, with cereal, this is rarely necessary.) Fruit continues to be added, a change being suggested at 5 or 6 day intervals, in case allergic manifestations occur.

21 DAYS

Meat.—At 3 weeks, the infant is started on the infant meats at the 2 p.m. feeding. My preference is for the Swift infant meats as they are pure and not mixed with other meats. My practice is to start with beef liver, then go on to pork, beef heart, lamb, beef, veal, and then fish, now that certain types are available in this form. The reasons for alternating the beef are obvious. Again, one starts with one tea or one dessertspoonful, and the quantity is increased as the baby desires. The meat is made to suit the baby's taste. Since he has become accustomed to a sweet taste in everything he gets, the meat may be mixed with sugar, milk or water, or salt. At this time also, a final change is made in his formula and he is jumped by one or sometimes two stages, to whole milk.

Vegetables.—These are now added to his diet at the 2 p.m. feeding, and may include any vegetable which may be pureed or mashed. Potatoes, mashed or riced may now be given at any time. Again, changes are made at 5 or 6 day intervals. Care is taken to see that only one change is made at these intervals, as by now the infant is eating practically everything and the trend is gradual toward a 3 meal a day routine. At this time the infant will usually sleep through the night requiring only a bottle at 10 p.m. Invariably, by about 6 weeks, the infant is able to go through from 6 p.m. to 6 a.m. although this is not forced,

and if the infant wakens at 10 p.m. he is given a bottle.

It has often been observed that there is less trouble in getting the infant to take meat than there is in getting him to start on his Pablum or cereal. While there have been one or two instances of the Pablum having to be thinned or thickened, or corn syrup substituted for sugar, and vice versa, so far there has been no record of an infant having to be coaxed to take his meat. It may also be observed here, that no force or persuasion has ever been necessary to induce an infant to take solid foods.

Egg yolk.—As soon as the meats are well started, egg yolk, raw and beaten into the milk, soft boiled and mixed with the cereal or meat, or hard boiled and grated into the meat, is next given.

Egg white.—Egg white is given next, either in suspension in sterile water, a drop at first, increased by one drop daily, or a small piece of the parents' harder boiled egg white, increased at 3 to 4 day intervals, until the whole egg white is given. At this stage a whole egg is given, as such, 3 to 4 mornings per week, or is mixed in with the meat and vegetables at lunch time.

By this time the infant is eating everything. The less expensive meat and vegetables, or cereal and fruit etc., mixtures can be given. Many of my patients now feed the infant from their own table, mashing the vegetables and fruits which form part of their own meals. Many of them also now give the infant his milk in a cup or glass.

One result of this system is that pre-school children (and most of those brought up under this system are, of necessity, in this group) and the few children who are now going to school, who were pioneers in this scheme, are not feeding problems to their parents because they "don't like this" or they "don't like that". All of them are accustomed to all vegetables from an early age and accept them as naturally as they later accept ice-cream.

Since the introduction of this method of feeding to my practice, my calls to sick children have been reduced by about 75%, the occasional cold and the omnipresent infectious disease accounting for the majority of calls. There is a definite decrease in sore throats and tonsillitis. The children gain weight more quickly and are solid and chunky, not fat and flabby. In view of the increase in weight the vitamin D intake has been stepped up, to take care of possible increased bone growth. These children teethe with less trouble, although the occasional one has difficulty. Their teeth seem to require less fillings and extractions are fewer. Their physical development is superior in general.

As it is my practice to follow up my delivered infants and supervise their care for at least one month after delivery, it has been a relatively simple matter to get mothers to co-operate with me, and while there was some doubt for the first year or so, the results obtained by this system were so apparent that the routine has become well established. I have yet to find an infant, physically normal, who does not thrive on this routine, and comparisons by mothers, with previous infants fed under the archaic system of no meat until 9 or 10 months etc., have shown that these infants thrive better, sleep better, and are far less trouble to their mothers than were their brothers and sisters in their infancy.

I recently had occasion to advise one of my mothers, who was moving to Ottawa, to take her baby to a well-baby clinic until she had established contact with a family physician. At the clinic the baby was examined. At first doubt was cast on the mother's story and list of foods that the child was taking, with the remark "The baby will have ulcers!" This remark, I think, well exemplifies the lack of thought on the subject of infant feeding.

My one regret is that it has been impossible to keep detailed records showing Hgb., red cell counts and graphs of weight gain. In general practice it is not good medicine to subject patients to extra expense merely for experimental purposes. However, the results, in the persons of the children, speak for themselves, and the keynote is simplicity.

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RÉSUMÉ

Certains prétendent qu'à trois mois les aliments mous devraient être donnés au bébé qui peut les digérer alors qu'il n'en a pas encore besoin pour sa nutrition. Dans cet article une méthode plus rapide est préconisée. A partir de l'âge de 7 jours: Le jus d'orange est donné pur, sucré avec du sucre de table ou du sirop de table. Une demi cuillerée à thé par jour pour commencer, en augmentant d'une demie cuillérée à thé par jusqu'à ce qu'au moins trois onces soient données. La vitamine D, est ajoutée au jus d'orange en augmentant la dose d'une goutte par jour jusqu'à ce que la dose nécessaire soit atteinte. Les céréales sont données à l'âge de 7 jours,

une cuillérée à dessert au début, à 10 heures a.m. et 6 heures p.m.; la quantité étant augmentée au besoin. La formule consiste en 1.5 partie de lait pour 1 partie d'eau.

Vers 12 à 14 jours lorsque les céréales sont bien tolérées on y ajoute des fruits: bananes, pêches, prunes, poires écrasées ou sauce au pommes. La proportion de lait et d'eau est porté à 2 parties pour une.

A 21 jours la viande est commencée et donnée à la dose d'une c. à thé, à 2 heures p.m. Il est préférable de ne pas donner de mélanges de viandes, mais une seul sorte de viande à la fois. La quantité est augmentée suivant les désirs du bébé. On peut y ajouter du sucre. La formule est remaniée de sorte que bientôt le bébé ne prend que du lait pur. A cet âge aussi les légumes sont ajoutés au menu de 2 heures p.m., sous forme de purées. Les changements se font à tous les 5 ou 6 jours, mais un seul changement à la fois doit être fait. Il s'ensuit donc que le bébé mange presque de tout et prend trois repas par jour et un boire vers 10 heures p.m. Vers la sixième semaine la nuit du bébé ira de 6 heures p.m. à 6 heures a.m. à moins qu'il ne s'éveille vers 10 heures p.m., et alors on lui donnera un boire. Il est plus facile de faire accepter au bébé la viande que les céréales, de plus, point n'est besoin de force ni de persuasion pour lui faire prendre des aliments solides.

Aussitôt que le bébé s'est fait à la viande le jaune d'œuf lui est donné nature ou bouilli ou encore mêlé à la viande aux céréales ou battu dans du lait. Puis c'est au blanc d'œuf à être donné dans de l'eau boullie une goutte d'abord puis en augmentant d'une goutte par jour. On peut encore le donner boulli en commençant par un petit morceau et en augmentant tous les 3 ou 4 jours jusqu'à ce que tout le blanc soit dommé. Alors, le bébé mange de tout et on peut tirer son alimentation du menu familial et lui servir le lait dans un verre.

Comme résultat, ces bébés ne causent pas de problèmes du vue alimentation et tous sont habitués à toutes sortes de légumes qu'ils acceptent tout naturellement. Les appels faits pour enfants malades sont réduits de 75% et ne sont faits que pour des épisodes infectieux. Il y a diminution marquée d'amygdalites et maux de gorge. Les bébés gagnent rapidement en poids mais ne sont pas gros, leur dentition ne cause que peu d'ennuis en général et les caries sont plus rares. En général le développement physiques est meilleur.

THE POTENCY OF PURIFIED EXTRACTS OF VERATRUM VIRIDE SOLD IN CANADA*

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DURING the past few years there has been increased interest in the use of veratrum products for the treatment of hypertension. A number of pharmaceutical companies are marketing products reported to be purified extracts of veratrum viride. A number of different methods and standards are being used for the standardization of these products. It was deemed of interest to investigate the variations in potency between different lots of purified extracts of veratrum viride using a reference standard with properties similar to the lots assayed and the same assay procedure. This paper is a report of our findings.

METHOD

A method based on the hypotensive effect in roosters was used to determine the potency of each lot.[1] This assay design was patterned after that described for the assay of epinephrine by Noel.[2] It has been found in this laboratory to give reproducible results, and to agree fairly closely with Maison's dog hypotensive method[3] as may be observed from the comparative results shown in Table II. The roosters were prepared for the assay in a similar manner to that described in the U.S.P. XIV for the assay of pituitary extract (post. lobe).

The extraction and preparation of the tablet and powdered samples, with the exception of those listed under Brand No. 2, Table I, were done according to the method described by Maison.[3] Those lots listed as brand No. 2 were prepared in a similar way, but on the assumption that each tablet was equivalent to one mgm. of alkaloids while the extract prepared from Brands No. 1, 3, 4 and 5 respectively was prepared on a per mgm. basis as labelled.

The reference standard employed for these assays was a pooled lot of an alkaloidal extract used by one of the pharmaceutical companies as a reference standard. The dog and rooster assays were done at different times on different extracts prepared from the same lot of tablets.

RESULTS AND DISCUSSION

In Table I are shown the results of assays of different lots of several brands of tablets sold in Canada. Also the results of assays of a number of lots of powdered purified extracts obtained from different sources are included in this table. These powdered preparations are used for the manufacture of tablets and for other products containing veratrum alkaloids. The potency of these powdered preparations are quite different ranging in potency from 59 to 233% of the standard.

The respective weighted mean potencies for the different brands of Tablets are as follows: Brand No. 1, 65%; Brand No. 2, 85%; Brand No. 3, 104% and Brand No. 4, 50%. Brand No. 5, a solution, was found to be 74% of the standard. The potency ranges for these weighted mean estimates are shown in Table I. A chi-square test showed that there was a significant difference in potency between the lots assayed. For the tablet preparations these potency estimates ranged from 40% to 108% of the standard. It may be observed that the variation within some brands is more than others.

*From the Food and Drug Laboratories, Department of National Health and Welfare, Ottawa, Canada.

It is apparent from these results that there are differences in potency between different tablet preparations of purified extracts of veratrum viride sold on the Canadian market. Whether these differences are of such a magnitude to be of clinical importance is not known but it would seem important from a clinical standpoint in establishing a dosage for a particular brand that each lot be of uniform strength. Each manufacturer is no doubt endeavouring to market a product of uniform strength but when different methods of assay and standards are used significant differences in the strength of different lots are almost bound to occur.

Whether the purified extracts of veratrum viride will continue to be used only time will tell. Current research on these products would seem to favour the pure alkaloids but as long as the mixture of alkaloids are used every attempt possible should be made to market a product of uniform strength and this can best be accomplished at the present time by each company adopting a standard qualitatively similar to their own product and using a method of assay that has proved to give reproducible results.

TABLE I.

RESULTS OF ASSAYS OF DIFFERENT BRANDS OF PURIFIED EXTRACTS OF VERATRUM VIRIDE BY A ROOSTER HYPOTENSIVE METHOD

Brand No.	*Type of product*	*Potency % Std.*	*Confidence limits P=0.05*
1	Tablet	69	65 - 74
		64	57 - 73
		60	57 - 63
		77	60 - 98
		75	60 - 93
		64	54 - 75
		74	63 - 86
		64	58 - 72
		66	59 - 74
	Weighted Mean	65	63 - 67
2	Tablet	101	91 - 112
		100	92 - 109
		97	88 - 108
		62	57 - 67
		93	81 - 106
		67	54 - 83
	Weighted Mean	85	82 - 89
3	Tablet	100	94 - 106
		108	97 - 121
		106	100 - 113
	Weighted Mean	104	100 - 107
4	Tablet	65	59 - 71
		40	37 - 43
		50	47 - 53
	Weighted Mean	54	52 - 56
5	Solution	81	74 - 88
		70	65 - 75
	Weighted Mean	74	70 - 78
Different Brands	Powder	74	64 - 86
		59	56 - 62
		61	54 - 69
		104	98 - 110
		233	217 - 250
		97	90 - 103
		108	104 - 113

TABLE II.

COMPARISON OF POTENCY ESTIMATES OF PURIFIED EXTRACTS OF VERATRUM VIRIDE BY THE HYPOTENSIVE ROOSTER AND DOG METHODS

Type of product	*Sample No.*	*Potency found in terms of Std. % Confidence limits P=0.05* — *Rooster method*		*Dog method*		*% difference between rooster and dog methods*
Tablets	1	77	60 - 98	74	65 - 83	+6.8
	2	75	60 - 93	76	64 - 87	—1.3
	3	64	54 - 75	77	63 - 91	—16.9
	4	74	63 - 86	70	59 - 82	+5.7
	5	64	58 - 72	76	64 - 87	—15.8
	6	66	59 - 74	73	62 - 85	—9.6

SUMMARY

The results of assays on a number of brands of purified extracts of veratrum viride sold on the Canadian market are presented. Significant differences in potency were found in different manufactured lots of this product. The rooster and dog methods gave results in fairly close agreement for several of the lots assayed.

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MELÆNA IN THE NEWBORN INFANT

Adults must ingest 50 to 100 ml. of blood in order to form a tarry stool; in a controlled study of 24 normal healthy newborn infants the authors found that as little as 2.6 ml. of blood, when ingested, may cause a tarry stool. An interval of 17 hours may elapse between the ingestion of blood and the passing of the first tarry stool. In 3 infants melæna was noted in 7 hours after the ingestion of blood. The average time of the first tarry stool following the ingestion of blood was 10 hours while an average of 2 tarry stools were passed, 2.0 to 5.0 ml. of blood being introduced into the stomach by Levine tube.—Rozenfeld, I. H. and McGrath, J. R.: *J. Pediat.*, 40: 180, 1952.

Canad. M. A. J.
June 1953, vol. 68

UTERINE BLEEDING: FUNCTIONAL, ULCERATIVE, AND CONGESTIVE

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THE OBJECTS of presenting a new classification of uterine bleeding in this paper are threefold. First, to indicate the main causative factor of the bleeding in simple terms. Secondly, a new conception is offered in the principle of including all functional bleeding under the two main headings "withdrawal bleeding in health" and "withdrawal bleeding in disease". Finally, included under the latter, withdrawal bleeding resulting from a fixed pattern of ovarian function is defined and described as a clinical entity. Uterine bleeding in pregnant states and that due to blood dyscrasia are not discussed in this paper.

UTERINE BLEEDING

1. *Functional bleeding.* – (a) Withdrawal bleeding in health. (i) Menstruation; (ii) excessive bleeding at the menarche; (iii) irregular bleeding at the menopause. (b) Withdrawal bleeding in disease. (i) A prolonged œstrin effect; (ii) a prolonged progestin effect; (iii) a fixed pattern of ovarian function.

2. *Ulcerative bleeding.*–(a) Simple. (b) Malignant.

3. *Congestive bleeding.*–(a) Varicosities. (b) Mechanical: (i) adhesions (post-inflammatory states); (ii) tumours; (iii) misplacements.

FUNCTIONAL UTERINE BLEEDING

Functional uterine bleeding is the result of tissue and circulatory changes in the uterus which have been induced by the internal secretions of the ovary. The blood loss occurs when the concentration of ovarian secretions as supporting substances is reduced below a maintenance level, either by a lowered production or by local vascular conditions limiting supply to the tissues concerned. The bleeding is quite properly classified as "withdrawal bleeding" because of the removal of the hormone factor in tissue maintenance.

(a) *Withdrawal bleeding in health.* – (1) Menstruation. The blood loss which occurs at regular intervals of from twenty-six to thirty-four days, in temperate climates, following the regression of a mature ovarian follicle constitutes normal menstruation. It is withdrawal bleeding in health. The sanguineous discharge usually consists of laked blood. The tissue change in the endometrium is limited to desquamation of the functioning layer of mature secretory endometrium.[1]

During the build-up period before the onset of bleeding, the healthy patient experiences symptoms from water and salt retention in the form of a feeling of fullness in the lower abdomen and in the breasts. Mild headaches and minor emotional disturbances are not uncommon. The uterus contracts in a rhythmical manner which is interpreted as painful cramps in some patients, at the onset of bleeding. Included with the bleeding as a part of the withdrawal phenomena are the symptoms of temporary dehydration in the immediate postmenstrual phase, with loss of the feeling of fullness in the lower abdomen and in the breasts. The prodromal and the postmenstrual symptoms rarely require treatment in the perfectly healthy, the well nourished, the physically rested woman who is free from social and economic emotional upsets.

(2) Excessive bleeding at the menarche. Excessive bleeding at the menarche is characterized by profuse or prolonged bleeding with the first two or three periods. Cyclic prodromal symptoms of menstruation can be observed in healthy children before the appearance of a true menstrual period as a part of the withdrawal phenomena. It is not surprising that some perfectly healthy children will experience excessive uterine bleeding during the first two or three periods when desquamation of the functional layer of the endometrium becomes a part of the completed cycle. Excessive bleeding under these circumstances is best treated by conservative measures which will reduce the blood loss, such as reduced activity with rest in bed and replacement of the blood constituents by proper nourishment and iron supplementation when necessary. Replacement of blood transfusion is seldom urgently required.

(3) Irregular bleeding at the menopause. Irregular bleeding at the menopause is a variation in the normal cycle occurring at the onset of the ageing process in the ovary. It is caused by the failure of the follicular apparatus of the ovary to proceed through the developmental phases to the complete stage of maturation which precedes

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a full menstrual cycle. In some women the interval between the onset of the bleeding is twenty-four days or less. In others the interval between the periods of bleeding may vary considerably, eventually assuming the proportions of a six weeks or a longer period of amenorrhœa. It should be emphasized that there is an accompanying ageing process in the uterus as well as in the ovaries. In multipara particularly, the increase in fibro-muscular tissue resulting from frequent pregnancies in addition to arterio-sclerotic changes in the blood vessels are additional factors in continued bleeding. These changes appear to be aggravated by the low acyclic œstrogen stimulation prevailing at this age, and may become so pronounced as to warrant consideration in the category of diseased states.

The majority of women require no special treatment for bleeding during this healthy ageing process which affects the ovary, the other endocrine glands, and indeed, all tissues of the body in the mid span of life.

(b) *Withdrawal bleeding in disease.* (1) A prolonged œstrin effect. Uterine bleeding results as withdrawal bleeding from an endometrium which is maintained in the proliferative phase by ovaries in which the follicular apparatus does not proceed to complete maturation. Uterine bleeding from a prolonged œstrin phase in the endometrium can occur in temporary disease conditions during the adolescent and during the reproductive ages. This type of ovarian function resembles closely the type of ovarian function during ageing at the menopause. The effect on the endometrium is similar in the diseased state and in the ageing process. The uterine bleeding is unaccompanied by the prodromal symptoms which usually accompany normal menstruation. A small percentage of patients experience symptoms described as premenstrual tension. The ovarian dysfunction in these cases is secondary to a generalized disease state which is temporary. The ovary is not permanently damaged. The character of the bleeding is venous. Rarely is the bleeding so active as to suddenly affect the blood state, or to suddenly weaken the patient.

In the adolescent and during the reproductive age, treatment should be directed at the restoration of the general health. When health is restored the ovarian cycle which is normal for that patient returns.

(2) A prolonged progestin effect. Uterine bleeding occurs as withdrawal bleeding from an endometrium which is maintained in the functional or secretory phase by ovaries in which regression of matured follicular tissue fails temporarily or permanently. In the latter case lutein cysts or solid lutein bodies remain active in the ovary, at any period of the reproductive age.

Uterine bleeding from a prolonged progestin effect on the uterus is most commonly seen at either extremes of the reproductive age. It is more common in elderly multipara at the menopause than in nulliparous patients close to the menarche. When it occurs early in life, particularly if accompanied by precocity, granulosa-celled tumours of the ovary and adrenocortical hyperactivity must be ruled out.

Theoretically, the reason for the maintenance of the follicular apparatus for a time in the mature state, in the absence of a pregnancy, is that it is due to an imbalance between the pituitary gonadotropin production and ovarian hormone production at the two extremes of the child-bearing period.

These patients present symptoms from a physiological build-up which simulates an early pregnancy. There is detectable enlargement of the uterus with softening of the lower uterine segment. The bleeding is sudden, of hæmorrhagic proportions, it reduces the blood state suddenly and is frequently mistaken for the bleeding attending an early abortion.

The urgent treatment is to control the loss of blood and replace it by transfusion if necessary. In the adolescent gynæcological treatment should be conservative, whereas, in the elderly patient radical surgical measures should be considered.

(3) A fixed pattern of ovarian function. A fixed pattern of ovarian function is defined as a type of ovarian function which produces a response in the reproductive tract that cannot be improved permanently by any known treatment. The condition in the ovary may result from a hereditary developmental defect or from an acquired permanent damage. Clinically it is to be distinguished from the ovarian dysfunction in progressive endocrine disease and from the temporary ovarian dysfunction in disease described above—conditions which may respond well to adequate general and supplemental therapy.

The ovary is permanently damaged by severe states of metabolic upset from prolonged disease,

Canad. M. A. J.
June 1953, vol. 68

severe acute disease with involvement of the ovary or by prolonged avitaminosis with starvation nutritional intake levels. The damage to the ovary is variable, from complete atrophy, through all stages of attempted cyclic follicular development with resultant cystic disease of the ovary. The secondary effects on the uterus are correspondingly variable, from complete amenorrhœa with atrophic endometrium to irregular or cyclic bleeding similar to that experience in prolonged endometrial phases. Advanced maturation of the follicular apparatus with incidental extrusion of a healthy ovum is of rare occurrence, consequently uterine bleeding from a functional effect in the endometrium as in normal menstruation is uncommon in patients having a fixed pattern of ovarian function.

The symptoms of which the patient complains are progressive from the age in her life when the ovarian damage became effective. Beginning during childhood symptoms may run the whole gamut of gynæcological complaints from delayed sexual development through sexual behaviour problems, poor sex sense, irregular uterine bleeding, prolonged uterine bleeding, sexual frustrations, pelvic pain, late marriage, frigidity, dyspareunia, sterility to the emotional and the vasomotor disturbances of the menopausal type. The progression cannot be halted. Careful history taking and meticulous physical examination will usually elicit damage, parallel to the ovarian damage, resulting from faulty nutrition or disease in other tissues of the body.

The importance of recognizing a fixed pattern of ovarian function as a clinical entity is because of the progression of symptoms and signs which have the basis of origin in a state of ovarian deficiency. Hence ablation of ovarian tissue by surgery or radiation therapy is a very serious detrimental step in the treatment of these patients. Ovarian tissue should not be removed for simple enlargement or in attempts to alleviate indefinite pelvic pain.

ULCERATIVE UTERINE BLEEDING

(a) *Simple ulceration.*—Microscopic ulcerative lesions in atrophic endometrium and in endometrium which is maintained in a prolonged œstrin or progestin phase are a cause of uterine bleeding. When œstrogens are present in the blood and tissue fluids, these minute lesions tend to heal and the bleeding is controlled. The uterine bleeding may be described by the patient as being cyclic and it is only by carefully kept records that irregularity of the bleeding can be detected. Polyp formation in the endometrium is common under these circumstances. The bleeding in benign lesions of the cervix is due to simple ulceration.

(b) *Malignant ulceration.* — Ulceration may well be considered a late stage in the progress of malignant disease of the cervix and more particularly so in the endometrium. Very early malignant changes in the endometrium without ulceration must be present in advance of ulceration and bleeding.

Examination of tissues obtained by curettement in the case of the corpus uteri and by biopsy and cell smear in the case of the cervix is the only certain way to effect a positive diagnosis in ulcerative conditions.

CONGESTIVE UTERINE BLEEDING

Congestive uterine bleeding implies overfilling of the capillaries, the venous sinuses and the veins of the immedate drainage area of the uterus, with back flow of blood through open venous sinuses into the uterus. The degree of blood loss cannot be correlated with lesions in the endometrium.

(a) *Varicose veins.*—Varicose veins occur in the broad ligaments as a result of multiparity. Chronic engorgement of pelvic veins has been ascribe to excessive coitus and inordinate sexual stimulation. Irregular uterine bleeding resulting from such engorgement is classified as congestive uterine bleeding.

(b) *Mechanical.*—(1) Adhesions (post-inflammatory states). Uterine bleeding of the congestive type is frequently a troublesome symptom in post-inflammatory states. In many instances the ovaries have been damaged to the extent that no ovarian tissue capable of maintaining hormone levels which would promote endometrial tissue growth remains. Mechanical venous congestion is a prominent feature in the findings at operation. The uterine bleeding is from open venous sinuses which are usually concentrated above the internal os.

(2) Tumours. Tumours located in the lateral uterine wall or in the broad ligaments cause venous congestion with backflow of blood into the uterine cavity. The position of the tumour is of more significance as a factor in the cause of congestive bleeding than the size which it attains.

(3) Misplacements. Congestive uterine bleeding is associated with a percentage of misplacements in which there is an added mechanical factor such as impaction, descent or angulation and constriction of veins carrying return blood flow from dependent organs or tissues.

The treatment of congestive uterine bleeding is directed entirely at removing the mechanical cause of the overfilling of the veins. The method of treatment should be a suitable combination of postural therapy, application of proper supports, and elective surgery.

SUMMARY

This classification is presented as a part of a new method of teaching adopted by the authors in order to simplify the approach to the understanding of the etiology of uterine bleeding in women of all ages. The understanding of this subject in the past has been obscured by the use of classical terms such as oligomenorrhœa, hypomenorrhœa, metropathia hæmorrhagica, to mention only a few. To us in the light of modern knowledge these terms are cumbersome and obsolete. In recent times many classifications of uterine bleeding tend to increase confusion, lack completeness and are so complicated that they cannot be put to practical use by the clinician. It is hoped that this classification of uterine bleeding will prove to be inclusive and practical.

This paper is in the nature of a preliminary report. It is the intention to complete the etiology, the descriptions of the gross and microscopic pathological findings, and to discuss the treatment in detail, in subsequent communications.

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RÉSUMÉ

Une nouvelle classification des saignements utérins s'impose autant pour l'enseignement que pour faciliter leur traitement. Ce travail présente une classification des principaux facteurs causant le saignement et une nouvelle façon de classifier tous les saignements fonctionnels sous deux rubriques "Saignement par soustraction chez le sujet sain", et "saignement par soustraction chez le sujet malade".

(a) Saignement par soustraction chez la femme saine.

1. La menstruation est une perte de sang se produisant à tous les 26-34 jours par suite de la régression d'un follicule ovarien mur. Ce sang provient des lacs sanguins et le changement dans l'endomètre se limite à la desquamation de la couche fonctionnelle de l'endomètre rendu au stade sécrétoire.

Avant la menstruation, la femme a une sensation de plénitude ou de gonflement au niveau des seins et du bas ventre ce qui est dû à la rétention d'eau et de sel. Assez souvent il y a légères céphalées et troubles psychiques légers. L'utérus se contracte d'une façon rythmée, ce qui donne les cramps au début de la menstruation laquelle est suivie d'une déshydration temporaire. La femme normale aux points de vue physique et psychique ne requiert pas de traitement.

2. Le saignement excessif à la puberté est caractérisé par un saignement abondant et prolongé lors deux ou trois premières menstruations; ceci pouvant être dû à une prolifération de la muquese dans les quelques mois précédant les premières règles. Le traitement devra être conservateur en vue de réduire la perte de sang: repos au lit, nourriture abondante et fer. Les transfusions sont rarement nécessaires.

3. Le saignement irrégular à la ménopause est une variante du cycle normal se produisant au début de la dégénérescence de l'ovaire; il est dû à une incapacité du follicule ovarien de compléter sa maturation. Les cycles seront alors de 24 jours ou moins, ou encore de 6 semaines ou plus. Les multipares, à cause de l'augmentation du tissu fibro musculaire et de l'artériosclérose dans l'utérus auront plus tendance à ces changements. La plupart des femmes n'ont pas besoin de traitement.

(b) Saignement par soustraction chez la femme malade.

1. Effet estrogénique prolongé: Le saignement provient d'un endomètre maintenu dans la phase proliférative par des follicules qui ne mûrissent pas. Le saignement utérin dû à une phase estrogénique prolongée peut se produire pendant certaines maladies au cours de l'adolescence et de la période de reproduction. Ce type de fonction ovarienne ressemble beaucoup à celui de la ménopause, il n'est pas accompagné, ordinairement, des symptomes prodromiques habituels. Le malfonctionnement ovarien est secondaire à une maladie organique temporaire, et l'ovaire n'est pas atteint d'une façon permanente. Le saignement n'affecte que rarement l'état général de la patiente et ne provoque pas d'anémie. Il s'agit donc de traiter la maladie de la patiente et lorsque la santé est rétablie, le cycle menstruel habituel pour la patiente revient.

2. Effet lutéinique prolongé: Le saignement provient ici d'un endomètre maintenu dans la phase fonctionnelle ou sécrétoire par des ovaires dont les follicules mûrs ne régressent pas en temps. Quand cet état est permanent les kystes lutéiniques ou les corps lutéiniques restent actifs dans l'ovaire à toute période de l'âge de la reproduction. Ce saignement se rencontre surtout à l'adolescence et à la ménopause, et particulièrement chez les multipares rendues à la ménopause. Lorsqu'il se produit à l'adolescence, surtout s'il y a précocité, les tumeurs à cellules granuleuses et l'hyperactivité cortico surrénale doivent être éliminées. Il semble que le maintien du corps jaune soit dû à un dérèglement de l'équilibre entre les gonadotropines hypophysaires et les hormones ovariennes. Ces patientes présentent des symptômes qui simulent la grossesse au début. L'utérus est gros et son segment inférieur est mou. Le saignement est soudain et se rapproche de l'hémorragie, une anémie s'installe, et on peut la croire due a un saignement d'avortement. Il faut alors arrêter l'hémorragie et au besoin transfuser. Chez la femme à l'époque de la ménopause il faut envisager l'hystérectomie mais non chez l'adolescente.

3. Le mode fixe de fonction ovarienne est le type de fonction de l'ovaire produisant une réponse au niveau du tractus reproducteur qui ne peut être améliorée d'une façon permanente par aucun traitement. Elle provient d'un défaut héréditaire ou d'une lésion permanente au niveau de l'ovaire. On ne doit pas confondre cette affection avec une malfonction ovarienne se produisant au cours d'une maladie endocrinienne progressive ni avec un malfonctionnement ovarien temporaire au cours d'une maladie qui peuvent répondre au traitement.

L'ovaire peut être endommagé d'une façon permanente au cours de maladies graves avec atteinte de l'ovaire, ou au cours d'avitaminose avec malnutrition. La destruction ovarienne peut varier de l'atrophie complète jusqu'à l'atteinte minime, mais permanente qui n'entraîne que des troubles du développement folliculaire avec

maladie kystique de l'ovaire. Cette destruction amènera soit de l'aménorrhée compléte avec atrophie de l'endomêtre utérin soit des saignements irréguliers ou cycliques semblables à ceux que l'on rencontre dans les phases endométriales prolongées; ou encore l'on pourra observer tous les états intermédiaires. L'émission d'un ovule sain est très rare. Les symptômes varient avec l'âge et ont tendance à augmenter. Si l'atteinte s'est produite pendant l'enfance, on pourra observer un retard du développement sexuel, des problèmes de comportement sexuel, des saignements utérins irréguliers ou prolongés, des frustrations sexuelles, des douleurs dans le bassin, un marriage tardif, de la frigidité, de la dyspareunie, de la stérilité ou encore des troubles émotionnels et vasomoteurs tels qu'on les rencontre à la ménopause. La progression ne peut être arrêtée. Une bonne histoire de cas et un examen attentif révêleront parrallèlement à l'atteinte ovarienne, une atteinte résultant d'une malnutrition ou d'une maladie atteignant d'autres organes. L'ablation ou la radiothérapie du tissus ovarien est néfaste dans cette maladie.

Pour qu'il y ait saignement par congestion il faut que les capillaires les sinus veineux et les veines dans le voisinage utérin soient gorgés de sang avec un retour sanguin dans les sinus ouverts dans l'utérus. Les varices des veines des ligaments larges chez la multipare et l'engorgement chronique des veines du petit bassin par stimulation sexuelle excessive le produisent. Il peut aussi être dû à une cause mécanique: (1) Adhérences qui se produisent après les infections. (2) Les tumeurs situées dans la paroi utérine ou les ligaments larges. (3) La malposition telle que le prolapsus ou une mauvaise angulation. Comme traitement il faut donc lever l'obstacle par des supports ou la chirurgie. Les terms oligoménorrhée, hypoménorrhée et métropatie hémorragique sont désuets et ne devraient plus être employés.

50 CASES OF RESECTION FOR PULMONARY TUBERCULOSIS*

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MY FIRST WORD, if I may, will be to add to the title of this paper and say: our "first" fifty cases of resection for pulmonary tuberculosis. These are not presented as surgical feats, that era has long passed; but merely to see what practical lessons can be learned from them. And after all, resection for pulmonary tuberculosis is still young enough a surgical procedure to arouse some enthusiasm. All the cases presented today were operated upon between October 1951 and April 1952.

TABLE I.

TYPE OF OPERATION		
Total resections.....	16 cases.....	32% of series
Pneumonectomies.....	10 cases	
Pleuropneumonectomies	6 cases	
Sides: Left: 11 Right: 5		
Partial resections.....	34 cases.....	68% of series
Lobectomies.....	27 cases	
Lobe and a segment...	5 cases	
One or two segments...	2 cases	
Of these 50 cases: 27 were men		
23 were women		
Of these 50 cases: 33 were primary resections		
17 were post thoracoplasty		

This will show you the type of cases one deals with, when beginning this work in a sanatorium. There naturally exists some accumulation of material which must be sorted. This explains our high rate of total resections, 32% of the series, mostly all for destroyed lungs.

The incidence of lobectomies is also high, as compared to segmentals, 64% versus 4%. But with the development of segmental localization of lesions, and of our operative technique, this ratio has changed, as will be shown later. The apical segment of the lower lobe had to be resected in 17.6% of 34 partial resections. We think we might improve our results if we increased this percentage, and were a bit more exacting as to the quality of the apical segment left behind. Very often, we did not suspect the patient to have apical segment disease; and we consequently thought of the probable failure of therapy, had this patient been submitted to thoracoplasty.

TABLE II.

In our first 50 cases: 32% were pneumonectomies
64% were lobectomies
4% were upper lobe segmentals

This ratio is changing with time and experience as will be shown later.

In 17.6 of all partial resections, the apical segment of the lower lobe was grossly diseased and had to be resected.

None of these had had a thoracoplasty, consequently the involvement of this segment could not be blamed for the failure of the thoracoplasties in these series.

70 operations for 50 cases is more than we should like to have, but one must not stop after the first operation, if the future of the patient is at stake.

Empyemas.—One patient developed a tuberculous empyema, because, I believe, we did not remove the parietal pleura. She was submitted to thoracoplasty and drainage. She has done very

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well, did not develop a broncho-pleural fistula, and now has only a small discharging sinus. Her general condition is extremely good.

A second patient had a thoracoplasty because of a non-tuberculous empyema, this has now healed completely and the patient is doing well.

A third patient had an empyema consecutive to the development of a B.P.F. After the thoracoplasty the space dried and healed well, her B.P.F. is now closed and she is nearly ready to go home. We have not classified these patients in our good results.

Suspected B.P.F.s.—Three cases of upper lobectomy developed signs of possible B.P.F. Practically at once they were submitted to upper thoracoplasty. Two did very well. One had a reactivation on the other side following thoracoplasty.

TABLE III.

THE NUMBER OF OPERATIONS

50 cases were submitted to 70 operations.
10 cases were submitted to post-resection thoracoplasties:
- 3 because of empyema
- 3 because of suspected broncho-pleural fistula (B.P.F.)
- 2 because of proven B.P.F.
- 2 because of pain
- 2 cases had a second lobectomy
- 3 cases had a repair of their B.P.F.s
- 1 had a tracheotomy
- 4 had various superficial skin repairs

Proven B.P.F.s.—The first case had an upper and lower lobectomy. Foolishly we had preserved a large middle lobe. Both upper and lower lobe stumps opened widely. We then removed the remaining middle lobe, and amputated the main bronchus close to the carina. We should then have submitted the patient to thoracoplasty. We thought her general condition did not permit it. She developed, a few days later, a main bronchus B.P.F. and died shortly after.

A second B.P.F. after pneumonectomy was submitted to bronchoplasty as soon as the diagnosis was certain. The bronchial stump was dissected out, re-amputated and resutured. It also reopened in a few days. This patient was a post-thoracoplasty resection. His empyema space is draining well and we are treating his bronchial fistula with sodium hydroxide cauterizations. His general condition is very good.

A third B.P.F. also had a 'plasty of her bronchus. This being on the left side, the arch of the aorta had to be mobilized and the stump worked upon retro-aortically. The bronchus was reamputated and resutured. Immediately prior to this operation, while the patient was being turned, she flooded her bronchial tree, became cyanosed and developed cardiac standstill. The chest was immediately opened, cardiac massage begun along with bronchial aspiration and controlled oxygenation. Shortly all was restored to normal. During the actual repair, the endobronchial tube slipped out, and she developed exactly the same condition, the same treatment was applied, cardiac function was again restored and the operation completed. She died 6 hours after operation, after having given signs of normal activity.

One post lobectomy B.P.F. had a thoracoplasty, he infected his space and wound, closed his fistula and is now closing his wound normally.

A last B.P.F. also had a 'plasty and simultaneous thoracoplasty. The space drained for 4 months. Now both fistula and wound are well healed.

We believe in thoracoplasty and drainage following the appearance of a B.P.F. But if the patient is to be submitted to an operation, we also believe it worth while to do a plasty of the bronchus at the same time. We have not had much luck with the procedure, it is true, but if one was unfortunate enough to have to do many of those, a more precise and effective technique could surely be worked out.

Tracheotomy.—This procedure can be absolutely life-saving in the postoperative course. We have had to use it only once in this series, 4 days after pneumonectomy. The relief it gave a cyanotic, heavily secreting patient was dramatic.

THE MORTALITY RATE

The operative mortality was 6%. Two cases developed early B.P.F.'s and died as the immediate result of their condition.

One case of upper lobectomy was doing extremely well until about 6 days after operation. Then she developed a high fever and died. Post-mortem revealed a well healed bronchus, the absence of tuberculous activity, and a typical non-tuberculous broncho-pneumonia. It was later discovered that she had spent part of a previous January night in the bath room.

The case of late mortality had been submitted to a left upper lobectomy and segmental resection of the apical segment of the lower lobe.

At the same time, as he had a stenosis of the main bronchus, and as his lower lobe was normal, the stenosis was resected and the lower lobe anastomosed to the main bronchus. He did very well for about 4 months, then after a few small hæmoptyses, he had a severe one and died. Post-mortem revealed extensive arteriosclerosis, and rupture of the aorta into the raw surface of the lung.

Our total percentage of B.P.F.s is 12%. We believe we can lower this considerably, with time and experience. But one must remember that bronchial disease is not always visible bronchoscopically, and also, that certain patients seem to be deprived of the inherent quality of healing. It will not be easy to abolish totally the incidence of bronchopleural fistulas in the surgery of pulmonary tuberculosis.

TABLE IV.

IMMEDIATE RESULTS

1. *Mortality*
   Operative mortality (inside of 60 days after operation)
   3 of 50 cases . . . . Mortality rate 6%
   Late mortality (later than 60 days after operation)
   1 of 50 cases . . . . late mortality rate . . 2%
   Total mortality rate, 6 months to a year after operation: 8%
2. *Morbidity:*
   a *B.P.F.s:*

Early and proven	5 cases	10% of series
Post-lobectomy	2 cases	
Post-pneumonectomy	4 cases	
Late:	1 cases	2% of series
Total of series		12%

   80% of B.P.F.s were on the right
   40% died as a result of their B.P.F.s (two cases)
   60% are progressing well in spite of B.P.F.s
   0% suffered no ill effects from B.P.F.

   b *Empyemas:*

a With B.P.F.s	5 cases	
b Without B.P.F.	3 cases	
Total	8 cases	16% of series
c *Atelectasis*	4 cases	8% of series
d *Spreads of reactivations:*		
Fairly certain	5 cases	10% of series
Questionable	2 cases	2% of series
Total	7 cases	12% of series
e *Hæmorrhage*	2 cases	4% of series
f *Cardiac standstill*	1 cases	2% of series

We have not always found it easy to establish with certainty the diagnosis of broncho-pleural fistula. And we don't like to submit the patient to another operation until such a diagnosis is definitely proven. Some cases, a week post-operatively, have evacuated a small amount of blood-stained liquid, and that was the end of that. We feel that small pockets around the bronchus were thus being evacuated without the existence of a full fledged B.P.F. We don't want to hedge about the situation, and turn our eyes away from defeat, but further events proved that there was no fistula; we even looked for them with the bronchoscope. Today, we are gradually coming around to the idea that nearly every patient who does poorly postoperatively must have a broncho-pleural fistula. With this way of thinking, we believe we will find them earlier, and I don't think we will misdiagnose very often.

We do believe that a patient who develops a B.P.F. after a total resection should be treated as an emergency, the minute the diagnosis is certain. Ideally operation should be done before there is gross contamination of the pleural space, and before a state of toxæmia develops. The fistula should be repaired, a thoracoplasty done, and drainage instituted. But we do not believe that a patient developing a B.P.F. after a partial resection should immediately be submitted to a total resection. Most times there will only be a local empyema, well walled off from the rest of the lung, and this can be aspirated and sterilized daily. A small tailoring of the chest wall may help.

Our empyema rate will be lowered if we diagnose and treat our fistulas early and vigorously. Empyemas without fistulas are probably due to technical faults and these can probably be eliminated with time. Tuberculous empyemas should be prevented by doing pleuro-pneumonectomies when indicated, and by removing all possible tuberculous tissue.

We have had surprisingly few cases of atelectasis. In this respect, we believe immediate pre- and post-operative bronchial aspiration to be of importance. The rôle of physiotherapy can never be emphasized enough in these cases, as much for the immediate result as for the ultimate one.

Breathing exercises and coughing are begun the day of operation and insisted upon repeatedly for the first few days. A good preoperative lecture to the patient on the necessity of this has proven a great help. Partial resections all have double drainage with active suction. A negative pressure of between 10 and 20 cm. of water is maintained for the first 48 to 72 hours. Tubes are flushed twice a day to insure patency.

Spreads and reactivations. — We have not found it easy to differentiate between a spread and a reactivation. During this first series not all patients had tomograms. Spreads can be prevented, we believe, by using not only one but several safety measures. Medically the patient

is "dried" as much as possible before surgery. He then is aspirated on the table. A Carlens tube is used to separate the two lungs. Once the chest is opened, continuous suction is applied to the operative side, and complete collapse obtained. As little dissection as possible is done before the bronchus is clamped. In pneumonectomy cases at least, we believe the bronchus can be clamped and cut, between 1 to 10 minutes after the insertion of the rib spreader.

Should we have a spread in any of our cases now, we should feel very much inclined to blame either the anæsthetist or the surgeon.

To prevent reactivation, we insist that our cases do at least 6 months' sanatorium rest postoperatively. A few have left earlier, most are doing well. No general rule can be laid down. Each case must be evaluated on its own grounds.

TABLE V.

Present status of these cases 6 months to a year after operation.

4 are dead	Mortality rate	8%
46 are alive	Survival rate	92%

Of these 46 cases:

42 are negative in sputum and gastric lavage.
91.3% of living
84% of whole series

4 are positive:
2 in sputum
1—Was a reactivation or spread
1—Is case of late B.P.F.
2 in gastric lavage only

Results based on clinical and laboratory data:

Excellent	78%
Good	8%
Poor	14%

To date:

26% are back home reorganizing their lives
66% are still in sanatoria
8% are dead

Hæmorrhage.—Two cases have bled postoperatively. One, as mentioned previously had a severe hæmoptysis and died. The other, after a right upper lobectomy bled in his space. It was not alarming at any time. We watched him carefully over a period of months. We now have the impression that the collection of blood has organized into a solid mass which now serves as a plombage. As mentioned before, the only case of cardiac standstill was in a difficult case of B.P.F. repair. We believe cardiac standstill to be intimately associated with anoxæmia, and consequently in most cases the result of inadequate technique.

46 of our 50 operated cases are alive today, *i.e.*, 6 months to a year after operation. 42 of these are negative, improving constantly, and as far as we can see now, on their way shortly to a normal life after anywhere from a year to 18 years of disease. 4 cases are positive: 2 are positive only in gastric lavage, it is too early to evaluate the result of their operation. We feel they will do well. One was a reactivation or spread, who is now in his 9th month of postoperative rest. His general condition leads us to believe that with time he will heal his other side.

The last positive case is our case of late B.P.F. after pneumonectomy. She did very well after operation, but pleural fluid contained tubercle bacilli. We consequently insisted more on bed rest and antibiotics. Instead she went home. Eight months after operation, she came back to us, a good medical case: B.P.F. on the operated side, and a massive spread on the other side. We are dealing with her with a surgical outlook and she is gaining ground. Her condition is nevertheless critical.

TABLE VI.

OPERATIVE TECHNIQUE FOR FIRST 50 CASES

a—Position on table:
Lateral position ..... 40 cases
Face down ..... 10 cases

b—Anæsthesia:
Endotracheal tube alone ..... 18 cases
Carlens tube ..... 11 cases
Endo T. and Thompson blocker ..... 11 cases
Endobronchial ..... 10 cases

c—Bronchial aspiration on the table pre, per, and post operatively.
d—Hilum dissection as early as possible
e—Pleuralization of bronchial stump in lobectomies, intercostal muscle grafts to stumps in pneumonectomies.

TECHNIQUE AS USED TO-DAY

a—Always the lateral position
b—Routine on the table pre. and post. operative bronchoscopic visulization and aspiration
c—Anæsthesia always with one of either:
Carlens tube
Endotracheal and Thompson blocker
d—Immediate clamping and section of bronchus
e—Simple closure of bronchus with pleuralization or muscle graft.

Being very strict in our criteria we have made this table. No patient was classed as excellent unless he was absolutely so. In the poor and good category, we feel that before long, we shall be able to extract a few cases and graduate them to the "excellent" class. Our high percentage of people still in sanatoria is easily understood, when one thinks that the first patient was operated upon only a year ago.

These figures represent our efforts as we were endeavouring to better our technique. A variety

Canad. M. A. J.
June 1953, vol. 68

of methods were tried in a variety of cases. None were really bad, but few we felt were insufficient and inadequate. The face-down position we tried in a varied ten cases. But we don't feel we need it now. With our present method we can deal quite safely and easily with the wettest of cases. Its only debatable value we find is for better breathing and better elimination of CO_2.

The choice of the anæsthetic agent is left to the anæsthetist. As a rule we don't use explosive mixtures as the diathermy machine is always in use. Each case is studied to determine which type of airway will be used. We use the Negus bronchoscope and battery for our routine inspections and aspirations. It affords a better view than any other we have ever seen.

This table shows the more or less standard technique we have come to use routinely. The lateral position is practically always used. We do as a routine, on the table, pre- and post-operative visualization and aspiration through

TABLE VII.

TYPE OF OPERATIONS		
1st 50 cases		*Next 25 cases*
32%	Pneumonectomies	32%
64%	Lobectomies	32%
4%	Segmentals	36%

the bronchoscope. This is done conjointly by the surgeon and anæsthetist.

Breathing is through either the Carlens tube or the Thomson blocker.

Carlens tube.—This is a double lumen tube which permits easy and free breathing of each lung separately. This gives us the quiet operative field we desire, a dry bronchus, as aspiration can be continuous. When doing segmentals the lung may be collapsed and inflated at will. Its insertion is very easy, the same as any endotracheal tube, although Carlens himself inserts it under local and laryngeal mirror vision. One soon finds the way of inserting it, in order that each tube may be in the right place.

Thompson Blocker.—This is a long double tube, one path is used for suction, the other to blow up a small silk balloon at the distal end. When blown this balloon fits very snugly against the bronchial walls. It is inserted under vision through the Negus bronchoscope. Once in place, the balloon is filled with sterile water, the guide wire pulled out, and the bronchoscope withdrawn. Then an ordinary endotracheal tube is inserted. Anæsthesia and ventilation are done with one lung, while continuous suction is applied to the other.

Once the chest is opened the bronchus is immediately isolated, clamped and cut. Allis forceps are applied to the bronchial stump assuring a temporary air proof closure. The rest of the hilum is dissected and dealt with and the lung removed. The bronchus is reamputated proximal to the clamps and closed with interrupted simple 000 silk stitches. The stump is pleuralized or covered with an intercostal muscle graft.

Since these cases were studied, 25 more cases have been done. The change in ratio of the different types of operation can be seen.

The percentage of total resections is the same. But we have come to realize that we were removing too much lung tissue. Also, that as a consequence of this we were left with residual spaces which sometimes called for tailoring of the chest. By better evaluation of the different upper lobe segments, we now usually leave behind the anterior segment, which so frequently is normal. Due to the normal hypertrophic compensation arising in the anterior segment and in the apical segment of the lower lobe, and to the shrinkage of the diseased segments, if only the diseased area is removed, there remains very little residual space.

In conclusion, may we say that we have heartily adopted pulmonary resection for tuberculosis. The key to success, we believe, rests in the choice of the patients and the time of operation. In this respect, we owe our sincere thanks to our medical staff for presenting us the cases at the opportune moment.

We have healed no tuberculosis, and shall heal none. The patient alone can do that. But we have rid him of an irreversibly damaged zone of lung, with its ever present menace over his future health.

PHYSIOLOGICAL KNOCK-KNEE

A slight degree of knock-knee is normal in children from 1 to 6 years of age—this is the physiological knock-knee. A reasonably accurate measurement may be obtained with the patient lying on his back, the knees are held together while the space between the medial malleoli is measured. The feet should be held at 90°, since a lesser or greater angle will change the distance between the medial malleoli. A child at 3 years has the greatest deviation, up to 3.5 cm. A simple corrective treatment is to add a wedge on the inner side of the shoe, varying from 1/8 to 3/16 inch, depending upon the amount of correction desired.—Geppert, T. V.: *Am. J. Dis. Child.*, 83: 154, 1952.

CAROB FLOUR IN THE TREATMENT OF DIARRHŒAL CONDITIONS IN INFANTS

PROF. DE LA BROQUERIE FORTIER, GEORGES LEBEL, M.D. and ANTONIN FRECHETTE, M.D., *Quebec*

DIARRHŒA is one of the commonest symptoms of infancy and early childhood, and is particularly prevalent in the first year. The etiologic factors involved are often mixed or obscure, but in many instances the cause of the diarrhœa may be either enteral or parenteral; and these are rapidly rectified by complex therapeutic procedures.

Digestive pathologic physiology so easily demonstrated in the infant's gastro-intestinal tract, has led us to believe that diarrhœal conditions of infancy are due to disorders of motility, secretion and absorption in either the stomach, small or large intestines.

Whether pathologic processes going on in the body and interfering with normal physiologic function, are the result of food intolerance or infection; and whether constitutional factors are added to these, along with faulty diet and systemic infection; it is felt that the final result will be a definite cleavage in the interrelation existing between intestinal motility on the one hand, and biochemical and absorption factors in the gastro-intestinal tract on the other. Other scientific elemental facts, such as enzyme deficiency, bacterial contamination of an otherwise un-polluted intestinal content, are continuously being brought forth, and definite progress shows itself frequently.[1]

The fluid lost in the stools may cause a depletion either in the intracellular or extra-cellular compartments; or the diarrhœa may have a direct bearing on the total fluid content of both. It is obvious that dehydration will be followed by modifications in the hydrogen concentration pattern. Laboratory investigation will show to what extent dehydration has affected the acid base balance: and will be a guide in determining the amount of liquid required for re-hydration. It is to be noted however that certain clinical signs will prove helpful in determining the magnitude of fluid depletion. For instance:[2]

(a) Extra-cellular dehydration is characterized by a depressed vascular tone, a small thready pulse, wrinkled skin, sunken eyes and fontanels, and hypotension.

(b) With intracellular dehydration, there is thirst, parched lips and tongue, increased nervous irritability, lethargy and coma.

In severe or prolonged diarrhœa, "toxicosis," dehydration is complete and nervous symptoms at their peak.

This brief résumé of the pathologic physiology of diarrhœal conditions of infancy, will emphasize the complex aspects of their treatment. This is attained by electrolyte replacement therapy given intravenously. Chemotherapy is suggested for infection; cardio-respiratory stimuli and tonics are also employed basically. These various procedures will not be discussed in detail at this time, although their usefulness is quite obvious. We wish however to discuss in this paper, the results which we obtained by the use of Carob flour.

CAROB FLOUR

Carob flour (*Cerotonia siliqua*) is derived from the fruit of the carob tree, which is found in some of the countries bordering the Mediterranean, as well as along the coast of Syria, in Palestine, Greece, Dalmatia, Spain and Portugal.[3]

Known for many generations the bean-shaped pod, when reduced to powder, has the following composition:

Fat	0.1 - 1.0%
Protein	3.2 - 5.6%
Soluble carbohydrate	45.0 - 60.0%
Pectin	1.5 - 1.5%
Cellulose	2.0 - 5.0%
Lignin, hemicellulose	22.0 - 25.0%
Starch	1.0 -
Minerals (Ash)	2.0 - 3.1%
Moisture	7.0 - 12.5%

I would like to point out that carob contains a limited amount of fat and protein; but on the other hand it is very rich in carbohydrate, of which fructose and sucrose are the most prominent. It is very rich in lignin and pectin, substances of high molecular weight. These account for the detoxicating properties of carob flour.

POWDERED AROBON

Arobon* is obtained by combining roasted carob flour and starch (Soldor). The mixture is then heated and dried. The composition of this powder is given in the following summary:[6]

Fat	1.5%
Protein	4.5%
Soluble carbohydrate	45.0%
Starch	13.5%
Minerals (Ash)	3.5%
Pectin	1.5%
Crude fibre	6.0%
Lignin (hemi-cellulose)	21.0%
Moisture	3.5%

100 gms. = 270 calories.

We now recognize, from the description of Martin du Pan (1945)[7] that carob flour, especially rich in lignin and pectin, has marked mechanical and detoxicating properties. It further acts as a buffer; and in a mechanical way, it adsorbs toxins and bacteria formed in the intestinal tract, and inhibits their formation. It has a healing action on the epithelial cells and restores normal colloidal equilibrium.

*The carob used in this study was graciously supplied to us in its commercial form "Arobon" by the Nestlé Co. Inc.

METHODS OF STUDY

It should be stated that for our present studies we used the same plan of treatment as the one used in 1949.[8] A 5% gruel of Carob flour (1 ounce) in rice water (20 ounces) to which was added 3 to 4 tablets of saccharin (15 to 20 grains). This was the only formula given to all our infants regardless of age or the severity of the diarrhœa, during 12, 24, or 36 first hours of treatment. After this period, 25% of the daily liquid given, was replaced by liquid buttermilk; and the other 75% of the liquid ration was given in the form of 5% Arobon, during the 2nd and 3rd days. On the following days, liquid buttermilk was increased; and on the 4th day it gradually replaced the Arobon to the extent of 50 to 75% of the feedings; and on the 6th to 8th days, the flour was discontinued, and buttermilk alone was used.

In mild cases of diarrhœa (about 20% of our cases) with no secondary dehydration, and moderate liquid stools, we used from the onset a diet comprising 25% liquid buttermilk and 75% gruel of 5% Arobon. This method of administering the formula did not on the whole improve the results.

CLINICAL FEATURES

Statistical data. — In the past three years (1950-1952) 271 infants with diarrhœa were treated by one of us* with Carob flour. Some 26 classes of patients were omitted from consideration, because the technique followed differed somewhat from the one described in this report. In the 245 remaining observations 8 among them received identical treatment for diarrhœa on two different occasions, thus bringing our statistics to 253 cases.

Our clinical research had for object not only to show the effectiveness of Arobon in the treatment of infantile diarrhœa; but also its value and notable freedom from side effects, in diarrhœal conditions of very small infants and even in the new-born. We fed mixtures of Carob flour to groups of children of all ages; see Table I.

Mortality rate.—Of the 253 subjects, 23 died, thus giving a 9.09% mortality rate. Of these 23 cases under consideration, 11 (or 47.8%) died during treatment. A closer scrutiny of these causes showed that only 7 could be attributed to diarrhœa, and these to a hyper-toxic form, known today as "toxicosis". It will at once be quite evident that left pulmonary atelectasis along with a partial atelectasis of the right lung, will cause death regardless of the fact that Carob flour has been used to combat the diarrhœa. Aspiration bronchopneumonia or bilateral suppurative mastoiditis are often complicated by diarrhœa in infants and children. In these cases, death may intervene long before Arobon, or for that matter, any other type of therapy, has had time to act. Also, we may state that only our

TABLE I.

Age	*Cases treated*	*%*	
< 1 month.....	41	16.2%	74.7%
1 to 3 months.....	93	36.8%	
3 to 6 months.....	55	21.7%	
6 to 12 months.....	53	20.9%	
12 to 18 months.....	8	3.2%	
18 months.....	3	1.2%	
Total.....	253	100%	

7 cases of "toxicosis", can disqualify Arobon as an effective agent, if we so desire, in combating the "metabolic disease" syndrome, known as toxicosis. Our mortality rate is thus shown to be 2.76%.

The other 12 cases died long after we had discontinued the use of Arobon. Diarrhœal symptoms had been effectively treated, but death could not be prevented in the following conditions:

4 cases of toxicosis
1 case of typhoid fever
2 cases of bronchopneumonia
2 cases of otomastoiditis
1 case of nephrosis
1 case of pulmonary atelectasis and hydronephrosis
1 case of cardiac malformation

The only real "failures" of Arobon to wipe out the diarrhœal conditions were the following:

CASE 1

Patient aged 2 months hospitalized 12-6-51, having 12 to 15 movements per day for the past two days. He was suffering from acute bilateral otitis media. He died following two days' treatment with Arobon. It was impossible to obtain "carob stools" during the treatment or before death.

CASE 2

Patient aged 6 months admitted 24-8-52 with bronchopneumonia. On 7-5-52, diarrhœal syndrome. On the 9th, he was given Arobon. On the same day, operated for subacute bilateral mastoiditis. Died on the 17th, and there were no formed stools.

CASE 3

Patient aged 4 months; hospitalized on 25-9-52 for hydrocephalus. On 5-10-52 was having 8 watery movements per day. There was weight loss of 1 lb. 4 ounces

* de la Broquerie Fortier.

in two days. From 7-10-52 to 16-10-52 was given Arobon. Stools became normal in consistency on 9th and 12th days.

ETIOLOGY

As the etiology of the diarrhœa has a bearing on the character and the gravity of its effect on the infant's general condition, we have grouped our observations according to whether abnormal bowel movements arose from nutritional, infectious or constitutional causes; we have also taken into consideration the age influence of our cases. Since the influence of infection manifests itself clinically in mild, moderately severe, and severe forms; it was our opinion that these should be further divided into two different categories: moderately severe infectious diarrhœas and toxicosis. The following Chart will illustrate the information we wish to convey.

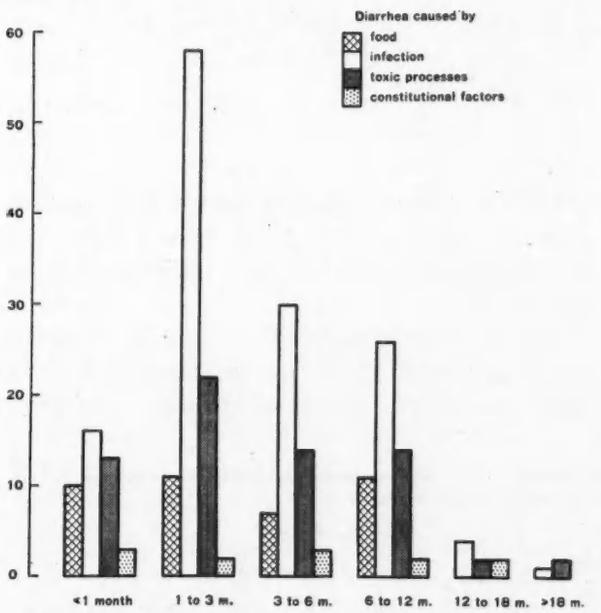

Chart 1.—Distribution of cases of diarrhœa according to age and etiology.

The following points are noteworthy:

1. The numerical importance of moderately infectious diarrhœal conditions, 53.47%.
2. The rather marked incidence of cases referred to as toxicosis; 33.8% of all diarrhœas having an infectious origin.
3. The exceedingly high incidence of infectious diarrhœas in the first three months of life.

It is noteworthy, that in our locality, the most frequent infectious cause seemed to originate in the middle ear. Acute otitis media, latent otomastoiditis were the conditions that gave rise to septic diarrhœas in over 50% of our cases.

Seasonal distribution.—There is evidence in the new-born to indicate that there are seasonal variations in the prevalence and severity of the disease. These are further subject to atmospheric changes during the various seasons of the year. We feel that a careful scrutiny of these seasonal variations in our cases of diarrhœa, will lend further validity to this assertion.

The month of August with 42 cases of diarrhœa, September with 30 cases, October with 33, were all considered to be the most favourable months for the occurrence of diarrhœal conditions (41%). It is general knowledge that atmospheric conditions, and the heat of August will produce variables in the intensity of the disease. Atmospheric variables occurring in October may also have a morbid influence, but at this period of the year, our interest centres far more on the type of animal feed; and in our opinion, this will constitute a far more important contributing factor. During this season, farm residue and dregs are used to feed our dairy cows. In many instances a careful study of these conditions would con-

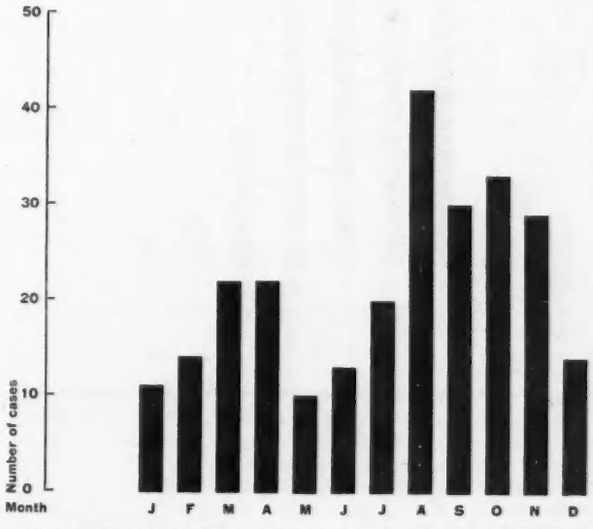

Chart 2.—Monthly incidence of diarrhœa, 260 cases, years 1950-51-52.

siderably reduce our cases of diarrhœa in the fall; and it is my opinion, that it would assist in placing our Provincial mortality rate on a parallel with, or lower than, infantile diarrhœa rate in the other Provinces.

CLINICAL RESULTS

On the basis of our experience, it seems to me that the real value of Carob flour lies in its prompt effect and regular action. This has been our objective in treating our diarrhœa patients. Table 3 shows that normal Arobon stools, dark brown in colour, homogeneous and firm, resembling goats' excrements, were obtained in

less than twelve hours in 35 infants. Stools were obtained in longer than twelve hours and less than twenty-four in 87 infants. In from two days to two and a half days in 68 cases. Thirty other cases in three days; in four days for 14 of our

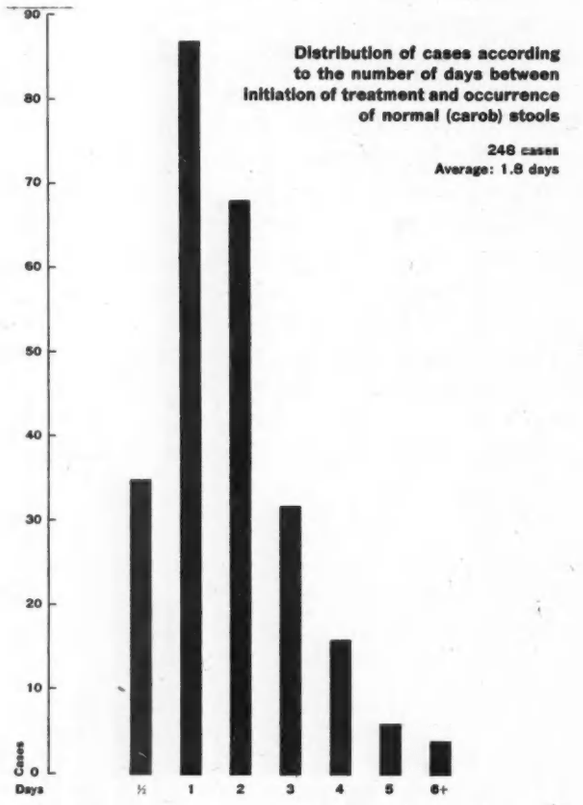

Chart 3.—The treatment of infantile diarrhœa with "Carob Flour".

normal Arobon stools in 36 hours. The etiological classification of our reports by no means modified these averages. This uniformity of action indeed, is another argument in favour of Carob flour's efficacy in checking infantile diarrhœa.

We do not wish to convey the impression that other effective therapeutic measures should be discounted, when normal Arobon stools have made their appearance. On the contrary, realimentation with a formula suited to the age of each infant is initiated, and during this period, the 5% Carob flour formula is rapidly diminished, and completely replaced in the diet by the 6th or 8th day. If from our observations, an attempt is made to determine the average time that Arobon was used, regardless of the amount given, for instance 2 teaspoonfuls per day, we find that the duration of therapy averaged from 6 to 8 days (Chart 3).

These results are similar to the ones given by other observers; and that we have ourselves reported in 1949.[9]

CONCLUSIONS

On the basis of our statistical findings and clinical studies, we believe that carob flour (Arobon Nestlé) is an effective agent in combating diarrhœa.

The prompt numerical reduction of bowel movements will offset the dehydrating effect of fluid loss with its biochemical results; obviating

TABLE II.

	Number of days of treatment with arobon															
Age	*1*	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*	*14*	*15*	*16*
< 1 month	0	1	3	3	6	9	5	4	1	4	2	1	0	1	1	1
1-3 months	0	3	1	6	6	22	9	4	5	7	5	2	1	3	3	8
3-6 months	3	2	1	8	4	11	3	6	5	1	4	0	3	1	0	2
6-12 months	0	5	7	10	3	7	6	6	1	1	1	0	1	0	1	3
12-18 months	0	0	0	2	2	2	0	1	0	0	0	0	0	0	0	0
> 18 months	0	0	1	0	0	0	0	1	0	0	0	0	0	0	0	0
Total	3	11	13	29	21	51	23	22	12	13	12	3	5	5	5	14

242 cases—Average: 7.2 days.
Average of "less than 11 days": 5.7.

infants; five days for six; and finally in six days and longer for four cases. In the 248 cases the average was 1:8 days.

The duration of Arobon flour formula showed no correlation with age curves. Loose, watery movements in half the number of our infants, became formed and dark-brown in colour in from 24 to 36 hours. More than 60% (63) of infants, whose ages averaged 1 month, obtained thereby the necessity of prolonged fluid and electrolyte replacement therapy.

The alarming and possibly catastrophic collapse seen during toxicosis is immediately corrected when this active agent is employed.

Its use in the new-born has shown the same absence of untoward effect that it has in older infants; being equally effective in both cases. It is particularly noteworthy that there is a

dramatic and rapid improvement within hours in severe toxic diarrhœas.

In the light of all our clinical findings, it is our opinion that carob flour is unexcelled by any other pectin basic medication that we have heretofore employed.

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ACTH IN THE TREATMENT OF KERATODERMIA BLENORRHAGICA

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KERATODERMIA BLENORRHAGICA is a rare disease, first described by Vidal in 1893 and characterized by dermatitis and arthritis secondary to gonorrhœa. When a disease manifests itself in divers systems of the body the most striking feature will often determine its classification. Hence, on reviewing the literature, it is noteworthy that few of the recorded case reports are to be found in journals of general medicine, the great majority appearing in journals of dermatology. As debility, anæmia, fever or polyarthritis may be the presenting symptom, as in the case to be reported here, this malady is of interest to general practitioners and internists as well as to dermatologists.

The employment of ACTH in the treatment of this disease is a modern, and apparently most effective, supplement to the therapeutic attack especially, as in this case, where resistance to antibiotics alone is encountered.

REPORT OF CASE

A nineteen year old girl was admitted to hospital on April 25, 1952. Early in January, 1952, she began to suffer from painful swellings of the feet. This complaint was severe enough to deter her from engaging in social activities, but did not prevent her from continuing her work as a waitress. During the latter part of January and through February her feet gradually became more swollen and painful. Towards the end of March she decided to apply poultices to the feet. After a few days of this treatment she noticed that the skin of the feet had become somewhat wrinkled and thickened and she was obliged to discontinue her work. It was about this time, at the end of March, that she developed chills and malaise and within two or three days other joints became swollen and painful, *viz.*, the knee, elbow, shoulder and temporomandibular joints. Very soon a spotty eruption appeared on the skin of the face, trunk and extremities. Anorexia and malaise developed and this, together with the severity of the joint pain and marked deterioration of her general condition, caused her to remain in bed.

The temperature on admission was 101°. The patient appeared very ill and in considerable distress. The mental state was one of apathy, dullness and defeat. Trismus, due to temporomandibular arthritis, was present and oral ulceration was severe. On the lower gums was a greyish, soggy, necrotic exudate and on the buccal and palatal mucosa reddish papules and grey ulcers, surrounded by erythematous areolæ, were present. The skin was pale and over the left cheek, the thighs and the trunk were small circular plaques, waxy and psoriasiform in appearance (see Fig. 1). The skin of the feet showed varying sized domeshaped conical vesicular-like lesions, which were firm, markedly thickened but not fluctuant (see Fig. 2). The above-mentioned joints were swollen, painful and tender, the ankle and knee joints showing more swelling than the wrists and elbows. There was a considerable effusion into the left knee joint.

The patient was confined to bed and symptomatic therapy was rendered pending diagnosis. Blood was transfused in volumes adequate to restore normal hæmic values. There was no abatement of the polyarthritis and the eruption spread progressively, so that it became confluent over the left cheek, around the ankles and over the soles of the feet where the skin became thickened and heaped-up. Elsewhere the individual spots enlarged. The scalp became severely involved and the hair became matted. The temperature continued to rise each evening to 101° for the first two weeks, to 100° during the third week and thereafter from 99 to 100° (during ten days of penicillin therapy) until May 29, 1952. Her general condition remained very poor until specific therapy was instituted on May 19.

Laboratory data.—Hb. 61%, W.B.C. 11,600, P. 73%, L. 15%, M. 9%, E. 3%. Sedimentation rate 38 mm. per hour. Hæmatocrit 33%. M.C.H.C. 26. The blood Kahn reaction was negative. The urine was clear. Three blood cultures were negative. Culture of joint fluid was negative. Spinal fluid analysis and Kahn reaction were negative. X-ray examination of the lungs showed no abnormality. Biopsy of the skin revealed in the epidermis a regular type of acanthosis with uniform elongations of the rete pegs and areas of intracellular œdema in the stratum spongiosum overlying the papillæ. A few neutrophils and lymphocytes were seen scattered throughout this layer and also in collections in the stratum corneum forming Munro abscesses. The stratum corneum showed a moderately thick layer of parakeratosis as well as fully keratinized cells intermingled with neutrophils. Histological sections (see Fig. 3) showed the presence of a spongioform pustule, with œdema and polymorphonuclear leucocytes situated in the upper layers of the stratum spongiosum and subcorneal layer. Other evidence of the psoriasiform picture was the thinning of the suprapapillary portions of the rete pegs and œdema and clubbing of the papillæ with capillary dilatation.

The cutis showed no reaction in the deeper portion but in the upper half, in the region of the papillary pegs and papillæ, there was œdema, capillary dilatation and a cellular infiltrate composed mainly of small round cells with some histiocytes and neutrophils along with occasional plasma cells. The histological picture was compatible with the diagnosis of keratodermia blenorrhagica. Cultures from the mouth grew out coagulase positive staphylococci. Cultures from the uterine cervix showed a heavy mixed growth with Neisseriæ gonorrhœæ on May 15.

Therapy.—Antibiotics were withheld pending diagnosis. With the isolation of gonococci penicillin, 500,000 units per day, was given. Within a day or two the patient noticed some relief from joint pain and slight increase

Canad. M. A. J.
June 1953, vol. 68

in joint mobility. After six days of treatment, however, this relative improvement was considered unsatisfactory and the temperature, while somewhat lowered, still ranged well above normal. Streptomycin, 1 gram per day, was added to the therapy, but after a further four days there was no appreciable effect from this additional therapy, although the skin had become somewhat less inflamed and the appetite was a little improved.

On May 29 (on the suggestion of B.K.) ACTH was administered, and continued in a daily dosage of 20 units, by slow intravenous drip over an eighteen hour period each day. Immediately there was a striking amelioration and regression of all symptoms. The temperature returned to normal as did the condition of the joints. The rash faded, desquamated and completely cleared leaving only a little pigmentation. ACTH was continued for ten days. After it was discontinued there was a very slight return of fever for five days. This then settled and after a further few days penicillin was discontinued. At the time of discharge on June 30 she could walk well, the feet were normal and all evidence of arthritis had cleared up.

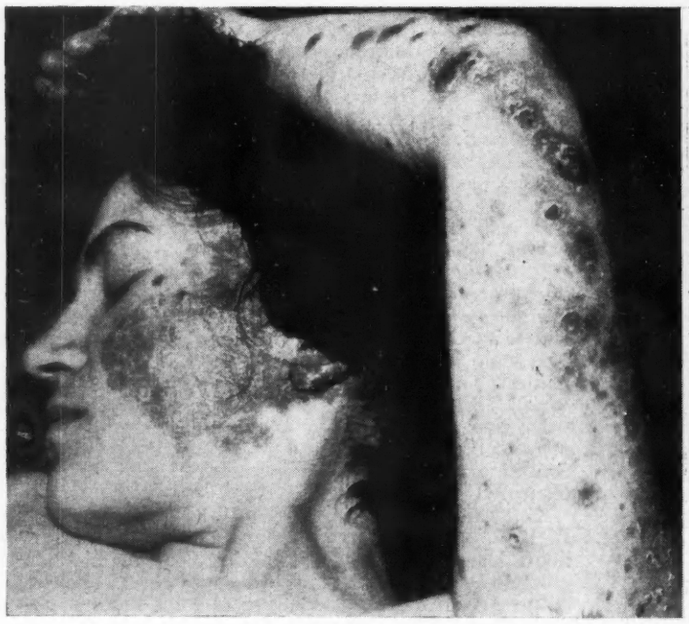

Fig. 1

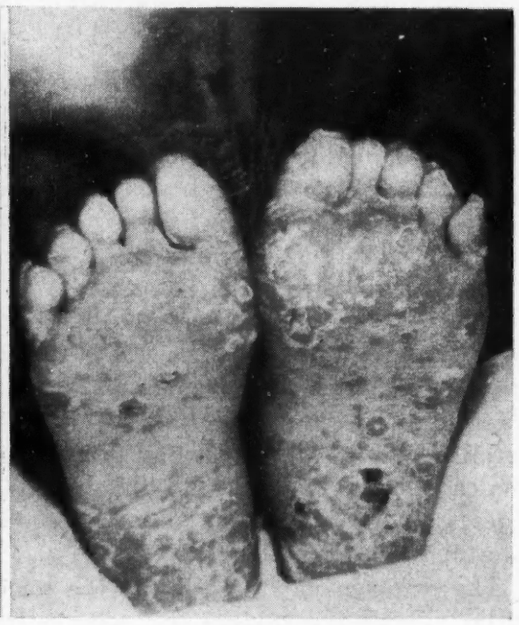

Fig. 2

Fig. 1.—Showing hyperkeratotic and psoriasiform lesions of keratodermia blenorrhagica on the face, ear, hairline, arm and forearm. **Fig. 2.**—Showing classical conical hyperkeratotic lesions of keratodermia blenorrhagica on the site of predilection—the soles.

DISCUSSION

It is widely known that acute gonorrhœa may be complicated not uncommonly by arthritis, mono- or poly-articular. It is also to be appreciated that, although rarely, involvement of the skin and oral mucosa may result from this infection. Various types of eruption occur and these may be hæmorrhagic, vesicular or urticarial. When the skin lesions, however, are characterized by marked keratosis in association with arthritis this constitutes the syndrome here discussed. In this syndrome the skin lesions are usually seen in patients presenting few symptoms of the disease but always during the course of the arthritis and nearly always limited to the legs and feet. It has been observed that this skin picture occurs during the chronic stage of the infection and rarely during a first attack. The sites of predilection are the internal plantar margins and dorsum of the great toe. At times lesions can occur on the head, trunk and extremities as evidenced by our case. Destruction or ulceration of the skin, however, never occurs.

The incidence of keratodermia blenorrhagica has been estimated to be as low as 1 in 4,000[1] to 1 in 7,500[2] cases of gonorrhœa while the incidence of gonorrhœal rheumatism is in the

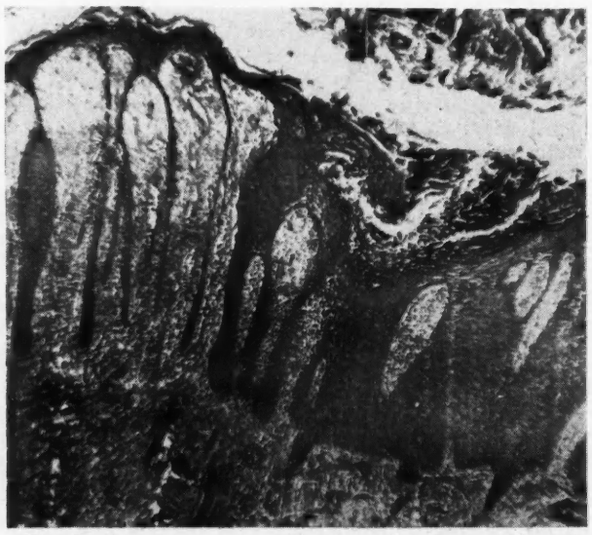

Fig. 3.—Low power magnification of lesion on the sole showing the regular acanthosis, with elongation of the rete pegs, thinning of the suprapapillary plates and capillary dilatation plus clubbing of the capillary waves and the presence of a spongioform pustule.

vicinity of 1 in 50 cases. The usual age period is the third and fourth decades. The male sex is affected about fifteen times as frequently as the female sex. Combes and Behrman[3] state that the syndrome "occurs almost exclusively in men in the third and fourth decade" while Ladany and Hughes[4] state that "the disease mainly affects the male sex, a curious fact never yet explained. We have found ten cases of keratodermia blenorrhagica affecting women and a ratio of 1 female to 15.3 males".

The pathogenesis of the disease has been the subject of much discussion and speculation. A good deal of overlapping in clinical description, and hence confusion, has existed between psoriasis arthropathica, Reiter's syndrome and keratodermia blenorrhagica. There are sufficient distinctive features to differentiate these three entities when they occur as typical cases but certain instances of Reiter's syndrome have so closely simulated keratodermia blenorrhagica as to make definite diagnosis very difficult and some authorities[5] consider that keratodermia blenorrhagica is Reiter's syndrome in which the urethritis proves to be specific rather than non-specific. Both these diseases are characterized by recurrences and both may undergo spontaneous resolution. For instance Lever quotes Kardung's case of Reiter's syndrome in which there were ten attacks while Freireich and Schwartz[2] record a case of keratodermia blenorrhagica in which there were four episodes between the years 1930 and 1943. Ocular manifestations may be found in keratodermia blenorrhagica and in Reiter's syndrome cutaneous lesions have been observed not too infrequently, for instance in 9 out of 45 cases.[5] On the other hand Reiter's syndrome is less constitutionally severe, there is less fever and no chills; it is self-limited (although it may continue for months) and it does not lead to permanent joint damage or to death as may be the case in keratodermia blenorrhagica. Furthermore, the isolation of Neisseriæ gonorrhœæ is not an accompaniment.

Pustular arthropathic psoriasis was simulated by some cases of keratodermia blenorrhagica and, in spite of the differences in the general systemic picture and clinical course, this in the past raised the question of the possibility of a common cause of the two diseases. Keim[6] (1924) pointed out that the eruption of psoriasis does not begin as vesicles and also that plasma cells are not seen in the infiltration. There are other clinical differences such as the site of eruption, psoriasis tending to appear over joints and keratodermia blenorrhagica to involve the skin of the feet.

Keratodermia blenorrhagica is associated usually with a protracted attack of gonorrhœa or with a past history of such an attack in most cases. In only about one-sixth of the reported cases has this not been so. In about 10% of cases there has been positive bacterial evidence as to the presence of gonococci in the cutaneous lesions[4] and in occasional cases in the blood and joint fluid, although it must be realized that in these fluids the organisms are notoriously hard to recover on account of the gonococcidal properties present. This seems to establish a positive etiological relationship but does not elucidate the genesis of the peculiar joint and skin reaction. It has been suggested that there may be a gonococcal septicæmia but this is a grave condition carrying a mortality rate of over 90% in the prepenicillin era. Nevertheless, there may be, and most probably is, a deposition of blood-borne organisms in the peripheral tissues which produce a direct effect. This leads up to another conception *viz.*, the toxic-allergic interpretation, which is supported by the presence of a syndrome, the symmetry and localization of the lesions, the difficulty in finding organisms and the coexistence of a primary focus.

The present consensus favours this functional allergic idea which postulates sensitization of the joints and certain areas of the skin and at times the conjunctivæ as a result of bacteræmia and by repeated deposition of organisms, or disintegrated products thereof, which act as antigens. This hypothesis would fit in well with the usual chronicity or longevity of the gonococcal infection in these cases. A bacterial allergic reaction on this basis may well declare itself with some violence and account for the resulting dramatic clinical picture with its fever, perspiration, heaping-up of corneous material in the skin and other marked systemic effects.

TREATMENT

As the identification and recognition of keratodermia blenorrhagica far outdates the discoveries of the various modern methods of treating gonorrhœa it is of considerable interest to review the case reports before and since their introduction and to note the impact of these

agents as regards therapeutic effectiveness. One is struck by the successes claimed respectively for treatment by fever therapy, the various sulfa drugs and penicillin as, in turn, they came into vogue. For instance Carr and Friedman[7] treated a patient who first had this syndrome in the years 1929-1932. After a remission of eight years he sustained a recurrence which was treated with sulfa and hyperthermia. These authors emphasize the uselessness of sulfa whereas Spencer[8] claimed complete success in a case by the use of sulfathiazole over as brief a period as ten days.

The advent of penicillin seemed to lend more promise as a weapon and apparently the first use of this agent must be accredited to Satulsky of Elizabeth, N.J., in January 1944. Here again the results of treatment lack uniformity for while in many cases they were dramatic or at least favourable[10] in some they were completely negative[9] in spite of adequate dosage. It must be fully appreciated that even without the aid of these remedies this disease would frequently undergo a natural, spontaneous remission and apparent cure, as indeed would many cases of uncomplicated gonococcal infection. Therefore claims of cure must be tempered by this appreciation along with the possibility of recurrence and therefore too much assurance must not be placed in any one remedy or regimen.

In the case herein reported, however, insofar as the abatement of the existing symptoms and clinical signs was concerned, the value of the adjuvant contribution of ACTH would appear to be incontrovertible. Undoubtedly the antibiotics, whether they be sulfa, penicillin, aureomycin, terramycin etc., are essential to cure the Neisserian infection but the addition of ACTH, especially in the intravenous form of administration, by its anti-inflammatory or antiphlogistic effect, dramatically reduces the morbidity of the otherwise long drawn out and disabling disease. In diseases manifested by marked inflammatory changes and which are self-limited or amenable to curative antibiotic measures, this dramatic response to ACTH is of particular value. It may be worthy of comment that the first clinical application of this combination of penicillin and ACTH in the treatment of keratodermic blenorrhagica was carried out by Myerson and Katzenstein in 1952.[11] Whether the result obtained in our case is indeed a cure or whether a recurrence will supervene, as has occurred in other cases, treated by other means or treated without any specific measures at all, will only be shown by a follow-up.

A follow-up report by her family physician Dr. A. A. Dixon, on November 17, reads as follows:

"The glabrous skin was entirely free except for very minimal post-inflammatory pigmentation of the legs and abdomen. There was no involvement of the palms or soles. As far as the arthritis is concerned, she stated in going up or down stairs there was some soreness of the left knee. I could elicit mild crepitations there. All in all, I would say she had excellent results from your therapy."

SUMMARY

1. Keratodermia blenorrhagica is a rare disease which complicates gonorrhœa and in which acute polyarthritis and constitutional manifestations are present and therefore is of interest to general practitioners and internists as well as to dermatologists.

2. The case reported is that of a young woman of nineteen years, which is unusual in that this disease is most often encountered in males and in the third and fourth decades of life.

3. The combination of ACTH with penicillin and streptomycin, after ten days of therapy with these drugs separately, caused an amazing recession of all the clinical features.

4. Further observations by different workers will be needed to assess the real value of this regimen, for the syndrome is rather too infrequent to be met with often enough by any one individual or group.

Appreciation is expressed to Dr. John Sturdy of St. Paul's Hospital and to Miss K. Hoskins of the X-ray Department of the Vancouver General Hospital for the illustrations.

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Canad. M. A. J.
June 1953, vol. 68

CORRELATION OF THE EOSINOPHIL COUNT WITH THE CLINICAL COURSE DURING CORTISONE AND ACTH THERAPY

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ALTERATIONS in the absolute level of the eosinophils were observed as long ago as 1910 by Bertelli,[1] but the importance of these fluctuations was not realized until Dalton and Selye[2] noticed that there was a marked fall in the eosinophil count during the "alarm reaction". Low eosinophil counts have been reported for several days postoperatively,[3, 4] in moribund patients,[5] in severe burns,[6] in the presence of severe emotion,[3] and in any condition producing a shock-like state such as a myocardial infarct.[7] Presumably the eosinopenia represents increased adrenal cortical function.

The gluco-corticoids are powerful agents in producing an eosinopenia—compound F being the most active followed by compounds E (cortisone) and A respectively.[8 to 11] ACTH causes a drop in the eosinophil count by stimulating the adrenal cortex.[8, 12] The way by which these hormones produce an eosinopenia has not been settled. A lysis of the eosinophils in the peripheral blood stream[13] has been suggested, but the majority of investigators[14 to 16] have not been able to demonstrate an intravascular destruction of eosinophils by cortisone or compound F, either *in vitro* or *in vivo*. It is possible that there is an interference with the passage of the eosinophils from the bone marrow into the peripheral blood stream. Rosenthal and his co-workers[17] found an increase in the marrow eosinophil count in all their patients treated with cortisone or ACTH, but Finch and his colleagues[18] found that during treatment with ACTH or cortisone, the marrow eosinophil count either increased or was not altered. A third possibility is that the bone marrow or the spleen or both may remove the eosinophils from the peripheral blood stream. The investigations of Finch[18] and Rosenthal[17] could be interpreted as supporting this theory but otherwise the evidence is unconvincing. Sevitt[6] and Luft[1] believe that the spleen, when stimulated by cortisone, traps eosinophils, but on the other hand, Durgin[19] and Speirs[8] deny that the spleen plays any part in this eosinopenia, citing numerous instances of the failure of splenectomy in altering the eosinopenic response to cortisone or ACTH. Godlowsky[13] gave heparin to a series of normal cases and found that if ACTH or cortisone were given, 10 minutes later, no eosinopenia occurred. He reasons that heparin mobilized eosinophils trapped in various organs.

The final possibility is that the bone marrow may cease to produce eosinophils and that since the eosinophil has a short life span a rapid fall in the peripheral eosinophil count will occur. Durgin[19] and his co-workers noted an increased proliferation of immature cells in the bone marrow during recovery from injections of lipo-adrenal cortical extracts. Possibly this may signify an increased output following decreased production of eosinophils. Goldman[17] found a decrease in the marrow eosinophil count in a case of periarteritis nodosa treated with ACTH. However, in view of the leucocytosis produced by ACTH or cortisone,[15] it is difficult to believe that, under these circumstances, the bone marrow selectively produces fewer eosinophils but more polymorphonuclears.

ACTH and cortisone do not always produce a fall in the eosinophil count. In diseases involving the bone marrow, with a low initial eosinophil count, ACTH and cortisone may lead to an increase in the number of eosinophils in the periphery due to improved hæmopoietic function.[3] Furthermore, if cortisone or ACTH is given for several weeks, the initial eosinopenia is often replaced by a steady rise in the eosinophil count which may reach higher levels than before treatment.[11, 18, 20]

A low eosinophil count (less than 30) is believed to indicate that the dose of ACTH or cortisone is satisfactory and that even if large amounts are given, no improvement in the patient's clinical condition can be expected. The many factors controlling the eosinophil count, make this assumption unwarranted. The normal range in any individual is wide. Rud[21] reports variations from 30 to 250 per c.mm. in 2,700 cases. He believes that 30 is the lower limit of normal, but Recant[9] would regard such a count as indicating an eosinopenia. Rud[21] has been able to demonstrate a diurnal variation in the eosinophil count. The level is highest in the early hours of the morning with a steady fall for several hours before noon, followed by a transi-

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Canad. M. A. J.
June 1953, vol. 68

tory rise and a longer fall until the evening and then a steady rise during the night. This pattern is independent of food intake or fasting,[22, 23] although it is possible that the latter produces an eosinopenia.[22] The diurnal pattern of the eosinophils can be altered by spontaneous "drops" to the extent of 50% of previous figures.[21, 23] These falls tend to occur in the mornings rather than in the afternoons.[23] The clinical condition of the patient may also affect the count, thus moribund patients will have a low count (less than 50) which may be unaffected by even large doses of cortisone or ACTH.

ACTH and cortisone differ in their effect on the eosinophils. The former is a stronger eosinopenic agent than is cortisone.[18] Several authors[11, 18] have noted that the eosinophil count in patients treated with cortisone tends to rise after the third or fourth week and to reach levels akin to or greater than before treatment. Robinson[11] and his colleagues believe that this effect cannot be prevented by increasing the dose of cortisone or if the eosinophils do fall again, it is transitory. Most authors have found that this "escape" or rise in the eosinophil count does not occur when ACTH is given.[11, 18] This statement may have to be revised in view of the report by Videboek[20] of an eosinophilia beginning about two weeks from the commencement of ACTH therapy in several patients with rheumatoid arthritis. The eosinophilia in these cases occurred only with some preparations of ACTH and was prevented by others. He concluded that the eosinophilia was due to a sensitivity to some products in the ACTH.

Allergic reactions to ACTH have been described[24] but to our knowledge, none have been attributed to cortisone and therefore, it is improbable that the eosinophil rise occurring later in cortisone therapy is due to an allergic mechanism. Robinson[11] suggests that the exogenous cortisone suppresses the production of endogenous eosinopenic agents produced by the adrenal cortex (cortisone and compound F). Since compound F is a stronger eosinopenic agent than cortisone, this would account for the greater effect of ACTH in producing a fall in the eosinophil count.

There have been several reports of serial eosinophil counts in a variety of disorders in an effort to relate changes in the count with clinical improvement or deterioration. Sevitt[6] followed 35 burn cases by repeated eosinophil counts. In those that succumbed, the eosinophils remained low, although in some of those that died, there were premature rises. Ellestad[7] noted that during the acute phase of shock following a myocardial infarct, the eosinophil counts were low and that the subsequent clinical improvement was accompanied by a rise in the eosinophil count. In a similar manner Jennings[5] observed 50 moribund patients. 94% showed eosinophil counts of under 30 just prior to death and in those that survived, the count rose to higher values, but the rise followed, rather than preceded, the clinical improvement.

PURPOSE AND MATERIALS

Patients treated with cortisone and ACTH were reviewed, in order to establish a relationship, if any, between a fall or a rise in the eosinophil count and clinical changes. The majority were patients suffering from rheumatoid arthritis or bronchial asthma.

All cases who had been submitted to operations were excluded, and those selected had symptoms and signs which could be assessed. The patients were divided into two groups: Group I included those treated before September 15, 1951, and Group II consisted of patients receiving cortisone or ACTH after that date. The reason for the division into two groups is that the eosinophil counts were not done at a fixed time before September 15, 1951. Since that time, they have been done at 1:30 p.m. after the patient has rested an hour. Capillary blood from a finger tip was taken at the patient's bedside, and the technique described by Randolf[25] was used in estimating the number of eosinophils. In Group I, the scale of daily cortisone was 300, 200, 150 and then 100 mgm. daily, whilst in Group II, it was 200, 150 and 100 mgm. daily. There were only four females in the two groups.

At least one control count was performed on each patient. On many ocasions, it was necessary to start treatment without delay, and consequently a control eosinophil count had to be taken at a time other than 1.30 p.m. Counts were done at least twice and more often three times weekly. Patients were considered to have shown a significant eosinopenia if the count fell by 50% or more of the highest control or pretreatment level, but this fall was not regarded as satisfactory unless confirmed by the next count. If the eosinophil count subsequently rose to a figure greater than 50% of the control level

for two or more consecutive counts, this was believed to be indicative of an "escape" or "drift" of the eosinophils.

DISCUSSION

In most of the patients, one control eosinophil count was taken, but eleven counts were performed on one patient in the forenoon and the range was from 656 to 287 per c.mm. This finding agrees with the "spontaneous" drops found by several investigators.[21, 23]

Normal counts were considered to be those between 100 and 500 per c.mm. Six patients in Group II had initially high counts (over 500) and in these an allergic mechanism played some part (one case of acute pemphigus and five cases of bronchial asthma). Five of them received cortisone intramuscularly, and all but one showed a satisfactory eosinophil drop. The remaining patient, who received ACTH, behaved similarly. Although a 50% drop occurred in the six patients, falls to below 100 were not found. One moribund patient had an initial count of 31, and oral cortisone caused a complete disappearance of the eosinophils from the peripheral blood field. In several other patients, isolated low control figures were obtained but the highest figure was always taken for the purposes of calculating a drop.

Two patients were given a course of cortisone followed immediately by a course of ACTH, and in each instance the ACTH produced a more marked drop in the eosinophils than did cortisone.

There is evidence that the eosinophils show a different response to two separate courses of cortisone. Two patients, given two courses of intramuscular cortisone separated by an interval of three weeks, showed a significant drop on the first occasion but not on the second, and one other patient not included showed a similar pattern, although a year elapsed between the two courses of cortisone (the first intramuscular and the second oral).

A significant drop in the eosinophils occurred in 70% of the patients in Group I and in 62% of Group II. It was felt that possibly the level of the control eosinophil count might be a factor in whether a drop occurred. In Group I, the average initial count for those that dropped was 302, and in those that did not drop, the average count was 122. On the other hand, Group II does not show this difference. The average count in those that dropped was 486, and in those that did not drop the average count was 539. It is therefore impossible to predict a drop on the basis of the control count.

In Group I, 14 patients showed a drop and

TABLE I.

CORRELATION OF EOSINOPHIL COUNTS TO CLINICAL COURSE
GROUP I. 22 PATIENTS TREATED WITH ORAL CORTISONE: EOSINOPHIL COUNTS TAKEN AT A FIXED TIME*

Immediate eosinophil response	*Immediate clinical response*	*Eosinophil change during treatment*	*Time escape occurred (average and range)*	*Duration of treatment (average and range)*	*Clinical course at time of escape*
Drop > 50% 14 patients	Improved..... 10	Escaped..... 9	9.1 days (3-23)	52 days (18-142)	Relapsed..... 5
					Improvement sustained..... 4
		No escape..... 1	—	37 days	Improvement sustained..... 1
	Not improved.. 4	Escaped..... 2	10.5 days (8-13)	23 days (17-29)	——
		No escape..... 2	—	15 days (12-18)	——
Drop < 50%	Improved..... 6	——	—	69 days (27-156)	——
8 patients	Not improved.. 2	——	—	18 days (10-26)	——

*1:30 p.m. after one hour's rest.

10 of them improved clinically. Two of these patients had diseases which do not respond to cortisone or ACTH. On the other hand, there were 8 patients in whom the eosinophil count did not drop, yet 6 of them showed a satisfactory clinical response. All but one of the 8 patients had diseases known to respond to ACTH or cortisone. In Group II, the picture is somewhat similar. There were 21 patients with satisfactory eosinophil drops and 12 of them improved clinically. Three of the 21 had diseases not known to respond to these drugs. The eosinophils did not drop significantly in 13 patients but 8 of these improved clinically and of the 5 which did not change, only one had a disease which does not benefit from ACTH or cortisone. Even discounting those patients having a disease not known to respond to treatment, it cannot be said that a drop in the eosinophile count parallels clinical improvement.

An "escape" in the eosinophils was not observed in a patient treated with ACTH for 33 days.

The majority of patients on cortisone in Group I showed a drift, but in Group II less than half developed an "escape". There is a discrepancy between the two groups regarding the time the escape developed. In Group I, the average was 9.8 days whilst in Group II, it was 13.3 days. No explanation can be given for this difference, but these figures are in accord with the findings of other observers.[11, 18]

The occurrence of an "escape" may be related to the duration of treatment. Usually it occurred between the 7th and 10th day of treatment, but in one patient it appeared on the 23rd day. Three patients in Group I did not escape. One of these patients was treated for 37 days, and the other two for 12 and 18 days respectively. In Group II 12 patients who had shown a satisfactory drop did not escape later. The average duration of treatment was 26½ days but the range was from 9 to 63 days. The lack of an escape or rise in the eosinophils in some patients may be due to the fact that treatment was not continued long enough for the eosinophils to manifest this change, but this interpretation cannot apply to all of them.

By the time the eosinophils began to rise, most of the patients in the two groups had been on 100 mgm. of cortisone for several days. Two patients in Group I developed an "escape" after they had been on 150 mgm. of oral cortisone for 7 days. In all the others, the dose was 100 or 50 mgm. The cases in these groups and others not included, tend to support the observation that this drift may be inevitable for some, and occurs even if the patients are kept continuously on

TABLE II.

CORRELATION OF EOSINOPHIL COUNTS TO CLINICAL COURSE
GROUP II. 34 PATIENTS TREATED WITH CORTISONE AND ACTH: EOSINOPHIL COUNTS AT DIFFERENT TIMES

Immediate eosinophil response	*Immediate clinical response*	*Eosinophil change during treatment*	*Time escape occurred (average and range)*	*Duration of treatment (average and range)*	*Clinical course at time of escape*	*Drug patients*
Drop > 50% 21 patients	Improved....12	Escaped.... 6	12.7 days (8-19)	29 days (16-49)	Relapsed... 3	IM cortisone... 3
					Improvement sustained. 3	IM cortisone... 3
		No escape.. 6	—	36 days (15-63)	Improvement sustained. 6	Oral cortisone.. 2 IM cortisone... 3 ACTH..... 1
	Not improved. 9	Escaped.... 3	14 days (5-23)	28 days (23-33)	—	IM cortisone... 3
		No escape.. 6	—	17 days (9-29)	—	Oral cortisone.. 2 IM cortisone... 4
Drop < 50% 13 patients	Improved.... 8	—	—	24.4 days (12-33)	—	Oral cortisone.. 2 IM cortisone... 5 Oral + IM.... 1
	Not improved. 5	—	—	22 days (14-28)	—	Oral cortisone.. 2 IM cortisone... 3

doses as high as 150 mgm. daily.[11] Of more importance is the fact that this "escape" does not indicate a deterioration in the clinical condition, and may occur in normal individuals. If the two groups are considered together, about half of the patients showing a drift deteriorated. Counts were not taken frequently enough to say whether the drift in the eosinophils preceded or followed the clinical change. Increasing the cortisone in those that had relapsed practically always caused a drop in the eosinophils and clinical improvement. But the increased dose was never maintained long enough to say whether these changes would have continued.

The mechanism of the eosinopenia produced by cortisone or ACTH has not been satisfactorily explained. One patient who had an aleukæmic leukæmia showed a marked increase in the WBC count when cortisone was administered. Previous to treatment, a single eosinophil count revealed no eosinophils in the peripheral field but whilst on cortisone, one count as high as 469 was obtained. This may indicate that cortisone does not inhibit the production of eosinophils by the bone marrow.

The authors believe that the initial response of the eosinophils to cortisone or ACTH depends in part on the sensitivity of the patient's particular eosinophils to gluco-corticoids. The drift or escape, occurring after the 7th day, may be due to suppression of the patient's adrenal cortex by the administered cortisone. This suppression, with a resultant decrease in the output of endogenous cortisone and more importantly endogenous compound F may be the cause of the subsequent escape of the eosinophils. Serial eosinophil counts might be of value in determining when this suppression occurs.

CONCLUSIONS

(1) A significant drop can be induced by cortisone or ACTH, irrespective of the magnitude of the initial eosinophil count. (2) ACTH is a stronger eosinopenic agent than cortisone. (3) If two courses of cortisone are given, the drop on the second occasion is less than on the first. (4) A significant drop in the eosinophils occurred in 70% of the cases in Group I, and in 62% of those in Group II. But the majority of patients improved clinically whether they showed an eosinophil drop or not. (5) An "escape" or "drift" in the eosinophils occurred as early as the 3rd or as late as the 23rd day of treatment with cortisone, but usually it was in the earlier part of the second week. No "escape" was observed in the patient receiving ACTH. The "escape" may represent the suppression of the adrenal cortex by cortisone administration. (6) This "escape" may be inevitable for some cases and probably cannot be prevented by giving large doses of cortisone (150 mgm. daily). (7) Of those cases showing an "escape" only about 50% deteriorated clinically. (8) No further light has been thrown on the mechanism of the eosinopenia. (9) Eosinophil counts during cortisone or ACTH therapy are of no value in estimating the effective dose.

The authors wish to express their thanks to Dr. J. S. Polson, for many of the case reports used in this study.

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GYNÆCOMASTIA DURING DIGITALIS THERAPY

Fourteen cases of gynæcomastia are reported occurring in men between the ages of 53 and 77. The condition developed after prolonged digitalis administration which is against liver disease being the etiological factor, although liver function tests were abnormal in six of seven patients tested. Histologic study of the breast was carried out in two patients and demonstrated an abnormal amount of tissue with striking fibroadenomatous change in one case. The digitalis aglycones have a structural similarity to the steroidal hormones and may undergo esterification in the liver with resultant œstrogenic effect. This may be enhanced by liver damage on the basis of chronic passive congestion.—LeWinn, E. B.: *New England J. Med.*, 248: 316, 1953.

Canad. M. A. J.
June 1953, vol. 68

MYSOLINE IN TREATMENT OF EPILEPTIC AND NON-EPILEPTIC PSYCHIATRIC PATIENTS*

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MYSOLINE† is a recent addition to the family of drugs recommended in the treatment of epilepsy. Some pharmacological aspects of this drug and its value in the control of major epilepsy were presented in a paper by Handley and Stewart.[1] The authors indicate that Mysoline's chemical structure is closely related to phenobarbitone, but its chemical properties and pharmacological effects are sufficiently different to put the drug in a separate category. Its value in the control of various types of epilepsy was recently discussed by Smith and McNaughton.[2]

This paper is concerned with the effects of Mysoline on epileptic states with social-psychiatric implications and psychiatric conditions with epileptic implications. The field of interest in this investigation was directed to (1) its usefulness in epileptic patients in whom a non-convulsive attack would manifest itself as a psychiatric condition ("psychomotor epilepsy", etc.); (2) its possible rôle in treating non-epileptic psychiatric patients with episodic manifestations in whom electroencephalographic abnormalities were found; (3) psychophysiological factors relevant to treatment of patients with this drug.

The study is concentrated on a small group of patients selected on this basis from a psychiatric hospital and out-patient setting, and is in the nature of a preliminary report. During the course of a year 18 patients suitable for this study were available. The patients fall into two groups, those with and those without clinical epilepsy. There were 10 cases in the former and 8 in the latter group. The observation period with Mysoline ranged from three months to one year.

A. PATIENTS WITH EPILEPSY

Most of the patients in the epileptic group had mixed types of seizures. The seizures were classified as major, minor and "psychomotor" seizures. The term major epilepsy is applied to the grand mal convulsion. The term minor epilepsy is used for "slight epileptic attacks in which impairment or loss of consciousness is the most prominent symptom".[3] Two of the patients with minor seizures fulfilled the criteria of Lennox[4] for "petit mal", having alternate spike and wave discharges of about 3 per second in the EEG. Seizures characterized by this EEG pattern are referred to as "petit mal" epilepsy, distinct from the minor epilepsies with other EEG manifestations. The term "Psychomotor seizure" was applied to epileptic attacks according to the usage of Gibbs:[5] "periods of confusion with incoordination and apparently purposeful movements". We are aware that the use of this term is somewhat controversial and moreover may cover different aspects of the epileptic syndrome.[6] However, the term is widely used and circumvents the uncertainties of sub-classification. The data on the epileptic group are given in Table I.

From an etiological point of view the group of epileptic patients is a heterogenous one. In six cases (Nos. 3, 4, 7, 8, 9 and 10) the seizures were of unknown origin. Two cases (Nos. 1 and 5) showed certain clinical features suggestive of an acquired cause, although none was demonstrated. One case (No. 6) developed the seizures following operation for frontal lobe abscess. One case of epilepsy (No. 2) was presumably due to birth injury. The duration of epilepsy in these cases ranged from 2 to 25 years.

The nature of the "psychomotor attacks" needs further clarification. A description of such attacks in the above cases follows.

CASE 1.

This patient experienced several types of attacks. She had frequent "flashes" of indefinable terror accompanied by a sickening feeling in the stomach, and sometimes a feeling of numbness in the right forearm and fingers. She also had momentary loss of consciousness during which she would mutter words or fragmented sentences and have chewing movements with her jaws lasting a few minutes and followed by confusion. Occasionally elaborate "dreamy states" occurred, one of which warrants a fuller description.

The patient was standing before a counter in a store with her daughter. Her husband left them a few minutes previously and was standing at another counter. The patient had suddenly a "strange feeling, like a dream", that her husband had on a cap with earflaps. She walked up to him and asked him, "Didn't you have a cap on?" At that moment she realized that her question was inappropriate, got very excited about it, burst out crying, felt nauseated and experienced the taste of onions, as if she were "belching from onions". In relating this incident three days later she was quite indignant at her "foolishness", as she knew at the time that her husband had never worn such a cap.

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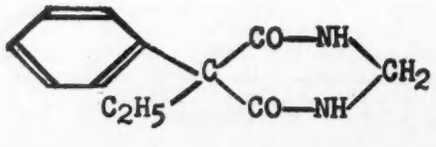
†"Mysoline" is the trade name of a drug developed by Imperial Chemical (Pharmaceuticals) Ltd. Its chemical name is 5-phenyl-5-ethyl-hexahydropyrimidine-4:6-dione and its formula is:



CASE 2.

The patient had short confusion states in which he laughed or cried for a few seconds and following one of which he was observed to pick up a pencil and start a cartoon, which he completed as he became more fully conscious. He was amnesic for the episode until half way through the drawing.

CASE 3.

The patient had attacks of automatic behaviour for which she was amnesic. They were related to her preceding activity, apparently purposeful, but inappropriate for the total situation. For instance, on one morning she was having a bath before going out. She experienced an aura as if the room were falling away, everything trembled, she was terrified and weak. She recalls pulling out the plug of the bath-tub and next thing she knew she was lying naked in her bed and the used towels were beside the bed and she was completely dry.

CASE 4.

This patient had attacks of automatic behaviour followed by amnesia. In one of these observed in the clinic he suddenly stopped talking, reached down to pluck at his shoe laces, straightened up again and began to apply paper clips to the crease of his pants. This behaviour lasted about 30 seconds, and when the patient became conscious he was astonished to find the paper clips in this strange situation.

CASE 5.

The patient had attacks of automatic behaviour followed by total or partial amnesia and, often but not always, by a convulsive seizure. Two attacks were observed in the hospital, one of which was described as follows:

The patient left her bed early at night (it is not known whether or not she was previously asleep) and she walked toward the nurse in a hesitant manner, apparently

TABLE I.

CLINICAL DATA ON PATIENTS WITH EPILEPSY

(1) *Initials, sex, age, and medication before* Mysoline*	(2) *Seizure frequency*		(3) *Side effects*	(4) *Psychiatric problems*	
	Before Mysoline therapy	*With Mysoline therapy*		*Before Mysoline therapy*	*With Mysoline therapy*
1. R.B. F., 51 yrs. Phenobarb (Dilantin).	Minor and/or psychomotor 1-2/day.	None.	"Grogginess" and occasional blurring of vision during first wk.	Depression, anxiety. Fearful and preoccupied about her spells. Unable to carry on household duties.	Some initial increase in anxiety. Active and happy in her household.
2. R.D. M., 37 yrs. Phenobarb, Dilantin.	Minor several/wk. Major 1-4/mo. Psychomotor 2-3/day.	Major 1/2 mos. Minor reduced. Psychomotor 2-3/wk.	None.	Slowness of thought and action. Inefficient at work leading to social deterioration.	Less slow, more efficient. Holds jobs longer.
3. D.R. F., 40 yrs. Phenobarb (Dillantin, Tridione).	Minor and/or psychomotor 1-2/mo. Major for several yrs.	Minor or psychomotor 1-2/2 mos. No major.	None.	Slow in thought and action. Paranoid tendencies. Unable to work.	Less slow. Paranoidal tendencies unchanged. Working full time.
4. J.W. M., 41 yrs. Phenobarb, Dilantin.	Psychomotor 25-30/mo. Major seizures controlled 5 yrs.	Psychomotor 10-15/mo. Major, none.	None.	Slowness in thought and action. Paranoidal tendencies.	Less slow. Tendency to euphoria. Paranoidal tendencies unchanged.
5. F.B. F., 43 yrs. None.	Major and/or psychomotor 1/1-2 mos.	None.	None.	Periodic drinking. Some depression. Unreliable.	Decrease in drinking. No depression. Unreliable.
6. C.H. M., 48 yrs. Phenobarb, Dilantin.	Major and minor 6-12 in 1½ yrs., controlled for 1½ yrs.	None.	None.	Frontal lobe syndrome. Slow, forgetful, little initiative. Paranoidal tendencies. Depressive.	No definite changes.
7. A.M. F., 14 yrs. None.	Psychomotor 1/2 wks. Minor 2/wk.	Psychomotor, reduction. Minor, none.	None.	Temper tantrums, unreliable.	No change.
8. B.J. F., 31 yrs. Phenobarb, Dilantin (Tridione, Mesatoin).	Major 8-12/yr. Controlled past 2 yrs. Petit mal, many per day.	Reduction of petit mal. No major.	Slight drowsiness. Morbilliform rash.	Labile mood, mostly depressive.	No change.
9. W.G. M., 16 yrs. Dilantin (Phenobarb).	Petit mal, many per day.	None observed.	Marked drowsiness and nausea.	Inefficient in school. Irritable.	Improved in school. Less irritable.
10. R.G. M., 24 yrs. Phenobarb.	Major, last one a yr. ago. Minor, 2/mo.	Major, controlled. Minor, none.	Dizziness and nausea for first 2 wks.	Inefficient at school, poor social adjustment.	No change.

*Drugs in ( ) were used in the past, and discountined for various reasons.

confused. Then she turned away, walked to the window, appeared to be looking out, although the blinds were drawn. Then she walked to another patient's bed, fumbled with the bedclothes for a few seconds, after which she walked towards the nurse again, dropped to the floor and developed a typical grand mal seizure.

CASE 7.

This patient had attacks of automatic behaviour for which she was amnesic. In one of these she was walking home from school and a few minutes later found herself at the end of a blind street some distance past her home. She also had episodes of violent behaviour, aroused with a minimal provocation, during which she experiences what she calls "pure rage". In this state she has been observed munching with clenching and unclenching of her fists. These episodes occur about every two weeks and are followed by a partial amnesia.

The phenomena which enabled us to group these attacks together and apply the term "psychomotor" may be seen from the above descriptions. It is not the purpose of this paper to go into a detailed analysis of the mechanisms involved in production of these seizures. However, it is worth indicating that all patients, except for Case 5, show in their EEG a concentration of abnormalities in the temporal area. Clinically, Case 1 shows most of the typical characteristics of a temporal lobe automatism and Case 7 shows one characteristic feature (munching). In other cases it is most likely that automatisms involving other areas are released during a minor attack.

Section 4, showing the psychiatric problems involved, requires further comment. It is evident that the psychiatric problems in epileptic patients arise from a complex system of factors. Most of them are psychodynamic in nature, active at least since the occurrence of the first seizure, and integrated thoroughly in the total personality. The evaluation of a drug over a relatively short period permits consideration only of those factors which operate on a more superficial level. For example, the short term impact of the change in frequency of seizures and the side effects of the drug-sedative or otherwise. It is inevitable that psychogenic factors will play a rôle and the changes observed are not necessarily due to the drug alone. The suggestive value of a new drug, and the increased attention given to the patients, tend to enhance the effectiveness. The validity of the psychiatric evaluation was improved to some extent by physiological testing, by the use of placebos, by changing back to the original medication for a few weeks, or stopping all medication for a time. This was done in such a way as to minimize the danger to the patient, and in the case of convulsive seizures changes in the medication were carried out under hospital conditions.

B. PATIENTS WITHOUT CLINICAL EPILEPSY

This second group of patients was selected on the basis of EEG and psychiatric considerations. All patients showed EEG abnormalities concentrated in the temporal lobe. The EEG findings closely correspond in Cases 11, 12, 17 and 18 with those which are called "epileptoid" in a paper by Rey, Pond and Evans.[6] Cases 13, 14, 15 and 16 correspond closely to the "constitutional" group described by the same workers. The common factor clinically in this group was the episodic nature of the symptoms. Other clinical features were various, and may be seen in Table II.

It is evident from the table that little or no beneficial effect was obtained in these patients with the use of Mysoline. In only 1 out of 8 cases was there a suggestion of clinical improvement. In this case there was some reduction of anxiety and decreased frequency in feelings of unreality. The same effect in this patient was obtained with dexedrine.

The distinguishing feature was the high incidence of side effects in these patients, as compared to the patients with clinical epilepsy. However it should be recognized that this group also had a higher degree of emotional instability and was less highly motivated than the epileptic patients.

DISCUSSION AND CONCLUSIONS

A preliminary evaluation of Mysoline based on the above case material is attempted in terms of (a) its usefulness in controlling seizures, (b) its limitations, (c) its impact upon the total personality and (d) its possible use in psychiatric patients outside the group of patients with clinical epilepsy.

(a) As for seizure control, our interest was focused on psychomator attacks, and the epileptic case material was selected mainly on this basis. Mysoline was found to give effective control in all of our cases with this type of seizure and proved to be superior to other drugs used in one of our patients (Case 1). With respect to grand mal attacks our experience was in keeping with the favourable findings of other investigators.[1, 2] In the two cases of petit mal

epilepsy our findings were very encouraging. One case (No. 9) was completely controlled, both subjectively, and objectively by EEG, as far as it can be determined from such a short period of observation. The other case (No. 8), with relatively small doses, showed a reduction of petit mal attacks subjectively, which was substantiated by EEG. Unfortunately, full doses could not be achieved as the patient developed a rash when an increase of the dosage was attempted.

The usual effective dose in epileptic cases ranged from 1.0 to 1.5 gm. daily (4 to 6 tablets). In one case (No. 4) an increase of the dosage to 1.75 gm. was well tolerated though insufficient for full control. In another case (No. 2) an increase to this dose was associated with side effects which necessitated a reduction to 1.5 gm. An effective control was best achieved in these two cases by a combination with Dilantin.

(b) With reference to the limitations to Mysoline therapy there were no toxic effects as evidenced by regular examinations of urine and qualitative and quantitative blood count. In one case (No. 8), Mysoline medication was discontinued, owing to development of a morbilliform rash. This occurred on increasing the dose gradually from 1.0 gm. to 1.5 gm. daily. Later the rash promptly recurred even when a single dose of 0.25 gm. was given.

Side effects occurred in the form of dizziness, drowsiness, nausea and occasionally vomiting. These arose during the first 24 hours of treatment and gradually disappeared in a few days, as a rule, with persistence of treatment. The side effects could be reduced to a minimum by gradu-

TABLE II.

CLINICAL DATA ON PATIENTS WITHOUT EPILEPSY

(1) *The patient and the clinical problem*	(2) *EEG findings*	(3) *Mysoline side effects*	(4) *Evaluation*
11. B.T. F., 18 yrs. Attacks of clouded consciousness with destructive behaviour. Episodes of depresonalization. Asocial, hypersensitive personality.	Excess random and paroxysmal 5-6/sec. activity bitemporally. Fast and slow alpha variants.	Dizziness, feeling "dopey" and nauseated for the first 10 days.	No change.
12. M.C. F., 27 yrs. Momentary attacks of "dream-like state" mainly feelings of unreality in a patient with mixed psychoneurosis (schizophrenia?).	Paroxysmal and random excess 4-6/sec. activity bitemporally and bifrontally.	Severe nausea, vomiting and fainting sensation with 0.75 gm. daily. Moderate nausea continued even with reduced dose.	Mysoline was not tolerated.
13. M.M. F., 15 yrs. Adolescent behaviour disorder with uncontrollable temper outbursts.	3-4/sec. and excess 4-6/sec. activity bitemporally. High voltage 3/sec. bursts in less than one minute of hyperventilation.	Dizziness and nausea for first few days.	No change with Mysoline. More relaxed and co-operative with Dexedrine, but had to be discontinued due to weight loss.
14. R.H. F., 25 yrs. Anxiety neurosis with feelings of unreality during panic reactions.	Excess 5-6/sec. activity bifrontally and bitemporally. Alpha variants of fast type.	Drowsiness, diplopia, blurred vision for first 7 days.	Some reduction of anxiety, less feeling of unreality with Mysoline. Patient noted similar effect with Dexedrine.
15. G.G. M., 22 yrs. Some stutter from 8 yrs. of age. Three "fainting spells" at age 18. Very anxious and at times depressed.	Excess 4-6/sec. slow waves chiefly from temporal areas.	Severe dizziness, nausea and vomiting for first few days.	No change in stutter. No change in mental state.
16. F.P. M., 29 yrs. Episodes of panic with crowds and in closed spaces for 5 yrs.	Excess 4-6/sec. waves bifrontally and bitemporally.	Dizziness and nausea. Sustained nystagmus persisted throughout. Mysoline discontinued.	No change in symptoms or personality.
17 A.L. F., 32 yrs. Episodes of panic, without apparent cause, for years. Occurred daily and lasted from minutes to hours.	Paroxysmal high voltage 6-7/sec. waves from all areas and sharp waves bitemporally.	Severe dizziness, nausea and vomiting plus drowsiness. Mysoline discontinued.	No change in symptoms or personality.
18. D.W. F., 25 yrs. A case of schizophrenia with episodic confusional states.	Occasional high voltage bursts of 4-6/sec. waves, most marked bitemporally.	Complaints of dizziness and "speeding up" of thinking. Continued effort intolerable to patient and she discontinued.	No beneficial effect. Acceleration of thinking processes may have been a true drug effect.

ally increasing with small doses (0.125 gm. b.i.d.). In spite of this, two cases, both in the non-epileptic group, were unable to tolerate the drug and terminated treatment.

(c) Favourable personality changes were seen in five patients (Nos. 1, 2, 3, 5 and 9). They showed an increase in alertness, drive and elevation of mood, accompanied by an improvement in their social adjustment. Psychological testing,* with tests involving speed of motor activity for both gross and fine movements, were administered to eight of these patients (Nos. 2, 3, 5, 6, 7, 8, 11, 13). Tapping, finger dexterity and digit symbol tests were included in the battery of tests. The tests were administered before treatment and later when the patient was stabilized on Mysoline. The results show a general increase in efficiency in the second performance, Case 3 showing the most significant gain. Two of the group (Nos. 5 and 13) show some decline on the second testing. Statistically valid conclusions cannot be drawn from the group performance because of the small sample and the lack of a control group.

It is difficult to be sure how much of this change was secondary to more effective seizure control and how much was a direct drug effect. We feel that perhaps the drug has some direct stimulating effect as seen in two of the non-epileptic patients (Nos. 14 and 18). This effect seems to be obtained with maintenance doses. On the other hand there was frequently initial drowsiness and in one case (No. 2) on maximum dosage, this feature returned. The apparently contradictory effects of stimulation and sedation is not yet understood but it is reminiscent of similar phenomena found with the action of alcohol. Some of the patients described their feelings as "grogginess" during the first few days of medication. The feeling was unpleasant, disappeared with maintenance doses, and no problem with addiction arose with our cases. Consequently individual patient reactions as well as drug dosage must be considered.

(d) Our non-epileptic group of patients were psychiatric cases selected on the basis described above. There was essentially no beneficial effect from treatment with Mysoline. However, in view of the mixed stimulative-sedative effect already mentioned, a further trial on other types of psychiatric cases might be warranted.

*We are indebted to Miss Jean Brown for adaptation and administration of these tests.

SUMMARY

The effect of Mysoline therapy was observed in 18 psychiatric patients selected on EEG and clinical basis; 10 were with and 8 without clinical epilepsy.

1. The drug provided effective control in all types of epilepsy studied, major, minor and psychomotor. There were 9 cases with mixed type of seizures, and 1 with pure petit mal attacks.

2. The usual effective dose in controlling epilepsy was 1 to 1.5 gm. daily. In two cases combination with other drugs was required: hydantoins were found preferable to barbiturates.

3. One patient on Mysoline developed a morbilliform rash, requiring cessation of medication.

4. No toxic effects were observed in bone marrow, liver and kidney function.

5. Initial side effects of dizziness, nausea and occasionally vomiting could usually be overcome by using graduated doses.

6. Five of the patients with epilepsy showed improvement of psychic and motor function and were better adjusted socially.

7. No beneficial effect was observed in a group of specially selected patients without clinical epilepsy.

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HEPATIC HYPOGLYCÆMIA: ITS OCCURRENCE IN CONGESTIVE HEART FAILURE

Impaired liver function is an important cause of spontaneous hypoglycæmia and episodic attacks may occur even when the liver damage is very mild and not clinically suspect. A group of 20 cases is reported, 13 having been autopsied and all demonstrating hepatic disease. In five of the cases the liver damage was on the basis of chronic passive congestion from cardiac failure. Attacks of spontaneous hypoglycæmia should be suspected in patients with chronic congestive failure since their symptoms may be erroneously attributed to cerebral anoxæmia. If clinical symptoms and signs suggest hypoglycæmia the blood sugar level should be checked and hypertonic glucose administered intravenously, as warranted.—Mellinkoff, S. M. and Tumulty, P. A.: *New England J. Med.*, 247: 745, 1952.

Canad. M. A. J.
June 1953, vol. 68

A CLASSIFICATION OF VARICOSE VEINS WITH A PLAN OF TREATMENT

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THE STATEMENT that a disease for which there are many remedies must still lack a curative one, is probably in no instance more applicable than to varicose veins. The word varix can mean an enlarged and tortuous vein, artery, or lymph vessel, and commonly we speak of varicose veins with the full implication of the derivation. However, the condition or conditions we so glibly label varicose veins, may, in many instances, not fulfill the term varicose, as no tortuosity need be present; neither is marked dilatation noted in many cases of saphenous insufficiency where symptoms may be marked. It is because of this fact that the term "varicose veins" is not completely adequate, but since it is so firmly entrenched by usage, we should, in our own minds, expand its derived meaning to include all conditions in which reflex venous flow produces symptoms, whether or not actual tortuosity or marked dilatation exists; or failing this, seek a more anatomical terminology for these venous insufficiencies.

It is not proposed to review the history of varicose veins in this paper, because it is most ably given by R. Rowden Foote[1] in his monograph on Varicose Veins; nor is it proposed to discuss the varied treatment procedures that at one time or other, have enjoyed a measure of popularity if not success in all instances. It is proposed to submit a practical symptomatic classification, which, it is hoped, will add greater understanding in the evaluation of varicose veins, and hence achievement of better results in treatment; and in addition to give support to procedures that have been the most efficacious in the author's hands.

Some time ago it seemed obvious that the reason varicose veins, so effectively treated by one author with one method, did not show the same good results by a similar treatment method used by other authors, was because we have not paid sufficient attention to the degrees of severity of the process by individual critical assessment in each case, but have attempted to treat many related but not identical conditions, all freely termed varicose veins, with a more or less standard type of treatment. For this reason alone we were doomed to more failures of treatment than we expected. I do not mean to infer that there is a definite or persistent cure available for varicose veins in general, but I do believe strongly that with proper categorization we can more accurately evaluate the treatment we use, and can in certain categories expect and get more lasting relief from symptoms than heretofore. After all, in the majority of cases, the patient comes seeking aid because of troublesome symptoms or signs, rarely for æsthetic reasons, though this latter can occur.

For the purpose of this discussion this paper recognizes, as does Pratt,[2] three sources of reflex venous flow in the superficial veins of the lower extremity sufficient to produce the symptom complex we agree to call varicose veins: (1) Insufficient orad valves; (2) Incompetent perforating vessels from the deep venous system; (3) Vestigial arteriolar communications at any level, not large enough to produce venous pulsation with but rare exceptions. The reflex venous blood in any given case of symptomatic varicose veins may be due to one factor alone, or to all the factors mentioned, or to any combination of them.

In addition to these mechanical or direct etiological factors, one must not forget the secondary or associated contributing factors of heredity, age, sex and occupation; nor must we be unaware of the part played in the aggravation of varicose veins by pregnancy, flatfootedness, lymphatic disease, pelvic tumours other than produced by pregnancy, infections of lower extremities, prostatism, chronic cough and wounds. Only a very careful history and physical examination will allow the maximum appreciation of the primary and secondary factors to which the disease owes its existence.

The following classification has been found of great value, not only as a guide in the treatment of varicose veins, but as a yardstick for postoperative assessment.

Grade I varicose veins. — Lower extremity veins which are visibly or palpably enlarged or tortuous but which in themselves produce no distress symptoms, regardless of their duration or of their magnitude, are classified as Grade I; surgical treatment is ordinarily granted for æsthetic purposes only, the exception is for

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Canad. M. A. J.
June 1953, vol. 68

females with Grade I varicose veins during pregnancy. Since pregnancy is one of the great aggravators of the condition of varicose veins, compressive support during pregnancy, preferably in the form of properly tailored or fitted elastic stockings, is strongly recommended as a prophylactic measure against aggravation of the varicosities; the stockings should be worn continuously while the patient is up and about, from the second month of the pregnancy until the third month post-partum, if the maximum protection is to be afforded against aggravation of the varicose condition. Without such prophylactic measures, Grade I varicose veins may well progress to Grade II or even Grade III without spontaneous regression after termination of the pregnancy. In my experience, there is seldom indication for varicose vein surgery during pregnancy, and the majority of pregnant women with symptomatic varicose veins can be managed conservatively.

Grade II varicose veins.—Visibly or palpably enlarged lower extremity veins, with minimal symptoms of pain in the legs, calf tenderness, or fatiguability. Minimal œdema may or may not be present at end of day. Surgery is not mandatory for Grade II varicose veins, but is advised if early œdema is present. Otherwise, depending on the occupation and will of the patient, treatment may be conservative with compressive support, preferably well tailored or well fitted elastic stockings. If longer than knee-length stockings are required to provide relief of symptoms, surgery is strongly advised. In the case of Grade II varicose veins with early œdema, preoperatively all patients are placed at rest with leg elevation for a sufficiently long time to allow complete subsidence of the œdema, and then properly fitted with knee-length elastic stockings which are required to be worn from one to three months postoperatively. Where surgery is indicated, my choice of procedure is radical stripping, with or without multiple excisions.

Where the varicose condition is multiradicular, staged procedures are used as repeatedly as may be necessary to remove offending veins. In uni-linear varicose disease one-stage stripping is a very effective procedure, but not so in the multilinear or multi-radicular arrangements. (In this discussion, the suffix -linear applies to tributaries of the long saphenous vein which course from low in the leg to high in the thigh before joining the parent vein, whereas the suffix -radicular relates to the shorter tributaries, running in an oblique or transverse direction, usually joining the parent vein below the level of the knee; *i.e.*, these latter are not full length tributaries.)

Grade III varicose veins.—With any or all of the symptoms as in Grade II, but showing in addition evidence of stasis devitalization of the skin with varying degrees of pigmentation, eczema, skin atrophy and the purplish discoloration of the pre-ulcer state of the primary ulcer, associated with or without but usually with varying degrees of low leg œdema. Secondary ulcers, which occur in a site previously ulcerated, will usually have less of the purplish discoloration surrounding them and more of the brownish pigmentation.

Treatment for Grade III varicose veins is preferably surgical combined with supervised compressive support and patient education. As œdema is usually but not always present to a considerable degree in this group, preoperatively all patients are placed on bed rest with leg elevation. When the œdema level has subsided or is minimal, the patients are fitted properly with below-knee elastic stockings, which they are told they will wear a minimum of six months postoperatively, and in the more devitalized and œdematous cases, indefinitely if they wish to have the maximum relief of symptoms. Termination of the wearing of compressive support is effected only when the patient can be symptom-free without this support, regardless of the time interval following surgery. Again, radical stripping, with or without multiple excisions, is my operative procedure of choice.

Grade IV varicose veins.—Symptoms and signs as in previous grades, but with the presence of a definite skin ulceration. In this group, the ulcer should be either healed or controlled to the point of healthy granulation tissue with active marginal epithelialization. This may be secured by bed rest and elevation with fungicides and compressive support to the limb. Once the ulcer is healed or controlled, surgical treatment and further management is as for Grade III varicose veins. For economic reasons, many patients cannot afford long periods of bed rest to heal their ulcer, although healing is faster by this means. For these patients adequate ulcer control can be secured by proper compressive support over a longer period of time.

At the Queen Mary Veterans' Hospital we have been using an effective, though simple, compressive support which meets the ideals of this form of treatment, namely, being form-fitting throughout the period of its application, allowing adequate surface respiration and maintaining a sufficient degree of elasticity. A *firm* elasto-crepe bandage is impregnated with warm Unna's, or similar paste, by being rolled through a container of it and on to a tongue depressor to give support for handling. The impregnated elasto-crepe bandage is then applied from the base of the toes to the knee, after subsidence of œdema. Excess of paste is wiped off and a second dry *firm* elasto-crepe bandage is then applied from the base of the toes to the knee, adding to the compressive support and protecting the overlying clothing from the mess of Unna's paste.

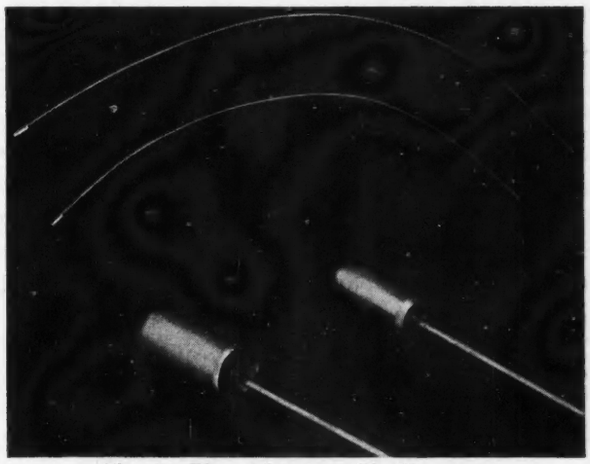

Fig. 1.—The strippers with the cone.

This outer layer will assume some of the paste from the impregnated layer. Such a compressive dressing, depending upon the activity of the patient concerned, can remain effective up to six weeks before replacement is necessary, usually because of soiling.

Grade V varicose veins.—Grades I to IV occurring in the post-phlebitic leg. In selected instances surgical procedures of a limited nature may be employed judiciously, but adequate relief of symptoms can be achieved in the majority of patients by custom-made full or knee-length elastic stockings and patient education.

THE STRIPPER

As the profession is aware numerous articles dealing with the stripping procedure for varicose veins mention, in each instance, a particular type of stripper which is not readily available through the surgical supply houses. For this reason a picture is presented, (Fig. 1), of my own stripper which, because of the reasons above mentioned, is home-made. It consists of an inverted cone on a flexible steel shaft, with a small bead on the leading point. Through the courtesy of Mr. George Laflamme, of Liquid Carbonic Canadian Corporation Limited, subsequent copies of this stripper in stainless steel and chromed varieties have become available to me. The stripper comes in two sizes, and the length of the shaft is especially designed to prevent the extraction of the long saphenous vein in its entirety in one manœuvre. In Grade III varicose veins, the trauma of complete retrograde stripping of the long saphenous vein in one manœuvre is often more than the superficial leg tissues will tolerate.

SUMMARY

VARICOSE VEINS

A Practical Classification	*Plan of Treatment*
GRADE I Visible veins, symptom free.	For æsthetic reasons only. Compressive support in pregnancy.
GRADE II Visible veins with minimal symptoms of pain, tenderness, fatiguability, may or may not have minimal ankle œdema, occurring at end of day.	High ligation with removal of veins except in well localized varicosities where local removal is done. Compressive support 3 to 6 months if œdema present. Compressive support in pregnancy.
GRADE III Grade II plus stasis devitalization, pigmentation, eczema, skin atrophy and varying degrees of œdema.	As in Grade II plus compressive support 6 months or longer.
GRADE IV Grade III plus ulcer.	Heal or control ulcer with rest, elevation, fungicides and compressive support. Then as in Grade III plus patient education.
GRADE V Grades I to IV in the post-phlebitic leg.	Custom made or well fitted knee or full length compressive support and patient education.

CONCLUSIONS

This paper emphasizes the need of critical specific evaluation of the stages of the condition we know as "varicose veins", and sets forth a simple categorization scheme with effective treatment procedures in each instance. The degree and duration of relief of symptoms avail-

Canad. M. A. J.
June 1953, vol. 68

able to the patient, with the treatment as outlined, is inversely proportional to the grading of the varicose veins as herein set forth.

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PSYCHIATRIC SERVICES IN GENERAL HOSPITALS IN CANADA*

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IN RECENT YEARS there has been growing recognition of the rôle of the general hospital in providing psychiatric services. Our present sources of information, however, as to the extent and type of service provided in Canadian hospitals have been incomplete and the need has been felt for more detailed data concerning these services. A survey of psychiatric facilities in general hospitals in the United States, completed in 1950 by the A. E. Bennett Neuropsychiatric Research Foundation, included reference to this development in Canadian hospitals.[1] But it was considered desirable to make a comprehensive survey of existing services in the larger general hospitals in Canada. During the early months of 1952 this survey was conducted jointly by the Research Division and the Mental Health Division of the Department of National Health and Welfare.

The survey was designed to obtain information at December 31, 1951 regarding Psychiatric Facilities and Admission Policy, Psychiatric Services and Treatment Provided, and the Movement of Psychiatric Patients in General Hospitals with more than 100 beds. With the exception of children's hospitals, chronic and special hospitals were not included in the survey.

Questionnaires were sent to 166 general hospitals and replies were received from 151, representing better than a 90% return. In view of the fact that only one follow-up letter was sent, the response was considered to be exceptionally good. A high proportion of replies was received uniformly from all regions.

CLASSIFICATION OF PSYCHIATRIC SERVICES

Of the 151 hospitals which returned the questionnaire, 73 (48%) reported some form of service for psychiatric patients. These hospitals may be considered as comprising four broad groups or classes according to the type and extent of service offered.

In the first group are 10 hospitals which have an organized clinical service in psychiatry, an active treatment program and a separate psychiatric unit of eight or more beds. This will be referred to as Class I.

A second group of 16 hospitals, Class II, has an organized clinical service but a more restricted active treatment program. Some hospitals in this group have a small unit of less than eight beds, but the majority have no definite bed allocation.

A further group, Class III, includes 40 hospitals which admit patients for limited treatment, diagnosis, or detention, but have no organized clinical service in psychiatry. Some of these hospitals have a staff psychiatrist or a consultant in psychiatry and limited treatment may be provided, but frequently patients are detained only until they can be transferred to some other institution.

TABLE I.

CLASSIFICATION OF GENERAL HOSPITALS BY PSYCHIATRIC SERVICES

	Number	*Percentage*
Class I	10	7
Class II	16	11
Class III	40	26
Class IV	7	4
Total with psychiatric services	73	48
No. of hospitals without services	78	52
Total replying to questionnaire	151	100

The final class is a small group, Class IV, which has psychiatric outpatient service but no inpatient facilities.

This grouping into four classes, as described above, is somewhat arbitrary but makes a convenient classification of psychiatric services provided and facilitates discussion and tabulation of the data. Table I gives the distribution of hospitals by class of psychiatric service.

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DISTRIBUTION OF PSYCHIATRIC SERVICES BY NUMBER OF BEDS IN HOSPITAL

Information on the distribution of psychiatric services by the number of beds in hospital is shown in Table II. In summary, some form of psychiatric service is provided by 83% of hospitals with more than 500 beds; 77% of hospitals with 250 to 500 beds; 45% of hospitals with 150 to 250 beds; and 20% of hospitals with 100 to 150 beds.

Of 24 general hospitals with 500 or more beds, seven have psychiatric wards or units and seven more have organized clinical services without segregation of psychiatric patients. Only four of these large hospitals have no service in psychiatry. Of the 30 hospitals with 250 to 500 beds, 23 offer psychiatric services of which three have a separate psychiatric ward. No psychiatric units were reported by hospitals with less than 250 beds. Of the 97 hospitals with 100 to 250 beds only three have organized clinical services in psychiatry.

TABLE II.

PSYCHIATRIC SERVICES BY NO. OF BEDS IN HOSPITAL

No. of beds	*Replies received*	*Hospitals with psychiatric service*					*Hospitals without service*
		Class I	*Class II*	*Class III*	*Class IV*	*Total*	
500 or more	24	7	7	5	1	20	4
250 to 500	30	3	6	11	3	23	7
150 to 250	42	0	2	14	3	19	23
100 to 150	55	0	1	10	0	11	44
Total	151	10	16	40	7	73	78

BED ALLOTMENT

Of the 73 hospitals which admit patients for psychiatric treatment, 28 have beds specifically allocated for psychiatric patients; 22 of these hospitals allocate a combined total of 318 beds while the remaining seven did not indicate the number of beds allocated. In the 10 hospitals with separate psychiatric units of eight or more beds, the psychiatric bed allotment reported is 255, or about 80% of the total.

TABLE III.

PSYCHIATRIC BED ALLOCATION IN GENERAL HOSPITALS

	Total bed capacity	*Psychiatric bed allotment*	*Percentage allotment*
Class I	7,789	255	3.3
Class II	7,837	25	0.3
Class III	9,775	38	0.4
Total	25,401	318	1.3

A comparison of the number of beds allocated for psychiatric patients with the total bed capacity in the same hospitals is shown in Table III.*

Psychiatric beds represent 3.3% of the total bed capacity in Class I hospitals. The allotment for psychiatric beds in all hospitals reporting some psychiatric service represents 1.3% of total bed capacity in these hospitals. In relation to the entire number of general hospital beds in

TABLE IV.

PROVISION FOR SPECIFIED FORMS OF PSYCHIATRIC TREATMENT

Type of treatment	*Class of hospital*				*Total hospitals*
	I	*II*	*III*	*IV*	
Psychotherapy	10	13	5	3	31
Sub-coma insulin	9	10	12	0	31
Insulin coma	7	5	7	0	19
E.C.T.	10	11	11	2	34
Total hospitals with treatment	10	14	19	5	48
Total hospitals with psychiatric service	10	16	40	7	73
Hospitals with treatment as % of those with psychiatric service	100%	87.5%	47.5%	71%	66%

*It should be remembered that the beds shown as allocated for psychiatric patients are less than the number actually used at any given time in as much as several hospitals do not allocate a definite number of beds for this purpose.

Canada, psychiatric beds constitute about 0.5% of the total.*

Treatment services.—The survey sought information on the use of four standard types of treatment—psychotherapy, subcoma insulin, insulin coma and electroconvulsive therapy. As shown in Table IV, insulin coma was employed generally less extensively than the other three. Also some variation was noted in the pattern of treatment according to the grouping of hospitals. In hospitals with organized services (Classes I and II) psychotherapy held a major place in treatment but the use of this therapy in hospitals with no organized services (Class III) was distinctly limited. In summary, some form of treatment is given in 48 (68%) of the 73 hospitals admitting psychiatric patients. Table IV also indicates the number and proportion of hospitals in each group providing treatment.

Psychiatric staff. — The services of qualified psychiatrists were available in 48 of the 73 hospitals providing some form of psychiatric service. However, as shown in Table V, less than half these hospitals engaged other specified psychiatric personnel including psychologists, social workers, psychiatric nurses and occupational therapists.

A qualified psychiatrist is in charge in all hospitals with an organized clinical service. The questionnaire did not attempt to secure information concerning arrangements by hospitals for the use of psychiatrists' services; the arrangements in some cases may be on a part-time basis and, again, more than one hospital may share the services of a psychiatrist. In some instances, psychiatrists are available only in a consulting capacity, particularly in hospitals having no organized clinical services.

Psychiatric units.—At the time of the survey, 10 hospitals reported separate psychiatric wards in operation, varying in size from eight to 43 beds and having a total bed allocation of 255. Closed and open wards appear in about equal number. Outpatient departments which are discussed more fully later are operated by eight hospitals with psychiatric units.

A fully qualified psychiatric staff, including a psychiatrist, psychiatric nurse, social worker, psychologist and occupational therapist is provided in four of the 10 units. In the other six units there appears to be no distinct shortage of any one type of psychiatric personnel; these units lack variously one or more members of a complete staff unit. In addition to the standard forms of treatment, the larger psychiatric units report the use of various other therapies such as nitrous oxide, ether and antabuse, group psychotherapy, narco-analysis and narco-synthesis, hydrotherapy, glissando shock and carbon dioxide therapy. One unit has an extension department for discharged patients and patients ready for discharge.

According to the returns received from the survey, six additional psychiatric units are presently under construction. These units will add about 100 psychiatric beds to the existing number. In addition, five other hospitals report that the establishment of psychiatric units is under active consideration. Hospitals which are constructing or considering psychiatric units include four with more than 500 beds and seven with less than 500 beds.

TABLE V.

HOSPITALS WITH PSYCHIATRIC STAFF

Psychiatric staff	*Class of hospital* I	II	III	IV	*Total*
Psychiatrists.....	10	16	15	7	48
Psychiatric nurses.	8	6	5	1	20
Social workers....	7	9	2	2	20
Psychologists.....	7	8	3	3	21
Occupational therapists.....	7	8	2	0	17
Total hospitals with some psychiatric staff....	10	16	17	7	50

Admission procedures.—In most hospitals admitting psychiatric patients, the admission procedures do not vary from those required for other diseases. In a few hospitals, as a matter of convenience, patients are admitted directly to the ward without passing through the admitting department and in several others approval for admission must be given by the head of the psychiatric service.

Day hospital service.—The "day hospital" was first introduced in 1946 as an experimental form of hospitalization for psychiatric patients at the Allan Memorial Institute of Psychiatry in Montreal.[2, 3] It was felt that psychiatric treatment could be furnished more effectively and less expensively to certain types of patients if contact with the home environment was retained. Under this system, patients enter the hospital for treatment during the day and return to their homes

*A total of 61,056 general hospital beds is derived from the Dominion Bureau of Statistics' Preliminary Report of Hospitals, 1950.

at night. This procedure is carried out until the termination of treatment.

A day centre, which is a composite unit taking care of both day patients and outpatients, has now been introduced at the Montreal General Hospital. Day patients receive various forms of treatment such as subcoma insulin, electro-convulsive therapy, narco-analysis, nitrous oxide therapy, individual and group therapy and occupational therapy.

A number of other hospitals, while not formally establishing day centres, have extended the period of treatment in the outpatient department to a point where the service offered is comparable in many ways to that available at the day hospitals.

OUTPATIENT DEPARTMENTS

Psychiatric outpatient departments are operated by 31 hospitals, about one-fifth of those reporting. Of these 24 are organized to provide both outpatient and inpatient services, while seven others operate only outpatient services. The majority of hospitals having outpatient services are those with organized clinical services for inpatients. Eight hospitals in Class I, 15 in Class II, one in Class III, and seven in Class IV have outpatient services.

Diagnostic services are provided in all psychiatric outpatient departments, but six do not give therapeutic service. A qualified psychiatrist directs each outpatient service and in 16 hospitals a psychologist is employed. Nursing care is directed by a clinical supervisor in psychiatry in 16 hospitals and social workers are employed in 14. Occupational therapy is provided in only seven. In 22 of the 31 outpatient departments psychotherapy is given; E.C.T. is provided at 17 hospitals, subcoma insulin at seven, and insulin coma at two hospitals.

Some variation is noted in the organization and the development of service provided by the seven hospitals which have an outpatient service only. In three hospitals, while service is centred in the outpatient clinic, consultation and treatment services are available to general hospital inpatients. In two hospitals the outpatient clinics are chiefly diagnostic.

Figures concerning the number of outpatients treated in 1951 are fairly complete. It may be pointed out, however, that the manner of recording caseload data by the hospitals varies somewhat; systems of reporting in some cases use the number treated while in others the total number of visits is used. A combined caseload of 6,874 outpatients was reported by 24 hospitals which gave data on numbers of patients treated. Twenty-five hospitals reported on the total number of visits by outpatients; there were 26,855 visits in these hospitals during 1951. Data derived from 21 hospitals which reported both numbers treated and the number of visits indicate an average of 4.2 visits per outpatient.

Psychiatric services in children's hospitals.—Questionnaires were sent to the six children's hospitals of more than 100 beds, five of which reported a psychiatric service. All five have psychiatric outpatient facilities and three have inpatient services as well. Those hospitals with inpatient facilities have a full staff complement and an active treatment program consisting mainly of individual and group psychotherapy.

TABLE VI.

PSYCHIATRIC PATIENTS UNDER CARE IN GENERAL HOSPITALS, 1951

Class of hospital	*Hospitals with psychiatric service*	*Hospitals reporting patients under care*	*Patients under care*
Class I.....	10	10	5,617
Class II.....	16	12	3,024
Class III.....	40	15	1,282
Class IV.....	7	0*	0*
Total.....	73	37	9,923

*Class IV hospitals do not have in-patient service.

The outpatient program includes both diagnosis and treatment in these five hospitals.

Volume of service.—While complete data on psychiatric patient movement was not available from all hospitals admitting psychiatric patients, data on number of patients under care were reported by all hospitals with psychiatric wards, most other hospitals with organized clinical services and several hospitals with very limited services. As shown in Table VI, approximately 10,000 psychiatric patients were reported under care during the year in 37 hospitals providing data.

Transfers to mental hospitals.—In the 30 hospitals which reported on both discharges and transfers, the average rate of transfer was 9%. Other relevant data are shown by class in Table VII.

Diagnoses of patients under care.—Reports on the diagnoses of inpatients under care indicate

that 27% of patients were psychotic and 73% non-psychotic. This compares with a rate for first admissions to mental hospitals in Canada of about 83% psychotic and 17% non-psychotic.[4] Psychoneurotics constitute the greatest number of patients (41.1%). They are followed by psychotics (27.1%), epileptics (8.1%) and mental defectives (5%). Further information on types of patients is shown in Table VIII.

Thirty-seven hospitals reported a total of approximately 10,000 inpatients under care in 1951 and 24 hospitals reported about 6,900 persons treated in outpatient departments.

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TABLE VII.

TRANSFERS TO MENTAL HOSPITALS, 1951

Class of hospital	*Number reporting*	*Discharges including deaths and transfers*	*Transfers to mental hospitals*	*Transfers as percentage of discharges*
Class I	9	4,358	430	9.9
Class II	11	2,481	174	7.0
Class III	10	550	61	11.1
Total	30	7,389	665	9.0

TABLE VIII.

PATIENTS UNDER CARE BY DIAGNOSES

Diagnoses	*Class I* Number	%	*Class II* Number	%	*Class III* Number	%	*Total*	%
Psychotics	1,029	29.8	592	19.7	463	37.3	2,084	27.1
Psycho-neurotics	1,550	44.8	1,116	37.2	497	40.0	3,163	41.1
Defectives	49	1.4	296	9.9	39	3.1	384	5.0
Epileptics	70	2.0	480	16.0	115	9.3	665	8.6
Others	761	22.0	515	17.2	128	10.3	1,404	18.2
Total patients by diagnoses	3,459	100.00	2,999	100.0	1,242	100.0	7,700	100.0
Number of hospitals reporting diagnoses and % of total in class	9	90.0	12	75.0	13	32.8	34	47.0

SUMMARY

Of 166 general hospitals with more than 100 beds to which questionnaires were sent, replies were received from 151. Seventy-three of these hospitals or 48% indicated the existence of some form of psychiatric service. Of approximately 61,000 general hospital beds in Canada, the total reported bed allotment for psychiatric patients in 1951 was 318 beds, representing 0.5%; 255 of these psychiatric beds were located in separate units.

Ten hospitals reported separate psychiatric units with eight or more beds. Six additional units were under construction in 1952 and five other hospitals were considering the establishment of units. Twenty-six hospitals reported an organized clinical service in psychiatry. Twenty-four hospitals operate both an inpatient and outpatient psychiatric service while seven hospitals are organized to give only outpatient service.

RATE OF GAIN AND SATIETY IN EARLY INFANCY

A consecutive series of 503 healthy infants born in hospital to 36¢ primiparæ and 125 multiparæ has been seen at a follow-up clinic during the second month of life, and the feeding history noted in detail. The rate of gain in weight for the whole group was determined for the period between the date of regaining birth weight and the first interview at the follow-up clinic. A group of 214 satisfied infants with an unblemished feeding history was obtained from the main series. The progress of the remainder (57% of the original series) was checked, usually by underfeeding, before the end of the second month. Sixty-eight per cent of the selected group gained 1.1 oz. or more daily compared with 49% of the remainder. The median rate for healthy infants in the first 2 months approaches 9 oz. per week. The optimal rate of gain for this age period is 16 oz. per week. The conception of a "stoking-up" process in the early weeks was suggested and exemplified by the weight curves of 3 healthy breast-fed infants. The standard formula for calculating infants' feeds is inappropriate for this age group. Objective evidence of the failure of orthodox methods was provided by studying satiation; only 57% of the formula-fed infants were satisfied with their feeds. A morbid fear of overfeeding, which was suspected in 26% of the mothers, is largely responsible for results which, from the infants' point of view, may permanently hamper their development.—Wickes, I. G.: *Arch. Dis. Child.*, 27: 449, 1952.

Canad. M. A. J.
June 1953, vol. 68

THE TREATMENT OF RAGWEED HAY FEVER*

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SPECIFIC DESENSITIZATION or hyposensitization in ragweed hay fever was introduced by Noon[1] in 1911 and further developed by Freeman[2] in this and subsequent years. Since that time the method has undergone minor variations but the main broad principles of treatment have remained unchanged. It is generally agreed among allergists that the method gives best symptomatic relief to ragweed sufferers and that, while a large proportion of untreated cases eventually develop seasonal or perennial asthma, specific desensitization reduces the number significantly.[3]

It is agreed that none of the antihistamine preparations should be relied upon solely for control of the average case of hay fever. There is, in fact, evidence to suggest that sole reliance upon antihistamines in treatment has led to the development of asthma toward the end of the hay fever season.[4] Their place is in the treatment of mild cases and as a supplement to hyposensitization when this is needed either for control of symptoms,[5, 6, 7] or given by simultaneous injection with ragweed for control of excessive reactions to the latter.[8] One advantage of their use in supplementing hay fever desensitization is their non-inhibiting effect on the formation of antibodies. They may, in fact, enhance antibody production occurring as a result of desensitizing injections.[9 to 12] It is difficult to choose among the antihistamines. A good deal of individual idiosyncrasy in tolerance of individual drugs is found to exist. In general, it is the experience of most persons who have prescribed these drugs, since the earlier ones appeared in 1945, that the most efficacious antihistamines are those with the most marked side effects.[13]

A single report only of the use of cortisone in hay fever is available. Stier and others[14] found that in 14 cases, ten of whom had both hay fever and asthma, six patients, though relieved of asthma, continued to have hay fever. Several patients requested return to desensitization therapy because of unpleasant side reactions; insomnia, headache, glycosuria, dysuria, acne and œdema. When satisfactory relief occurred, it was said to be of greater degree, however, than had been attained with any previous method of therapy employed. Treated cases were found to be sensitive to pollen in higher dilution after the season was over than they had been previously. Cortisone, it was concluded, is not satisfactory as a sole treatment for hay fever, but may be of value for brief periods of time at peaks of the pollen season, especially in cases with asthma.

Quantitative measurement of dosage has been facilitated by the use of the unit system. The pollen unit of Noon[1] was the original one introduced as the quantity of allergen contained in an extract of 0.000001 gm. of pollen. Objections to this were raised on the ground that a given weight of different samples of one species of pollen does not always contain the same quantity of the hay fever excitant. This, and a unit introduced by Coca based on total nitrogen content of the extract, have generally been supplanted by the protein nitrogen unit of Cook and Stull[15] in which one unit equals 0.0001 mgm. of protein nitrogen. Protein nitrogen content and activity of any extract do not always exactly correspond, but sufficiently so for practical purposes in fresh extracts.[16] Extracts are usually diluted in a weight by volume method so that 10,000 units are contained in 1 c.c. of a 1:100 solution. In the cases to be reviewed in this paper, the protein nitrogen unit has been the one employed throughout.

Of the three methods of hyposensitization, the co-seasonal is usually reserved for those who first present themselves for treatment during or shortly before the pollen season.[17] The method is purely empirical; dosage is low, usually two to ten protein nitrogen units although occasionally doses as high as two hundred protein nitrogen units have been used. There is wide variation among allergists as to the initial dose, the interval between doses and whether or not the initial dose should be repeated throughout or slowly increased with each dose throughout the course.[3] Vaughan[18] claims 60 to 80% relief, which compares favourably with 86% relief in preseasonal and 92% relief in perennially treated cases. In the pre-seasonal method, injections are started three or four months before the pollen season, gradually increased and then either stopped, or continued as a maintenance dose during the season, with the maximal dose achieved pre-seasonally or with a slightly re-

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duced dosage. In the perennial method, the highest tolerated dose achieved preseasonally is continued at two to four weekly intervals throughout the year. It is generally agreed that the perennial method of treatment gives overall best results.[19, 20, 21] Some find it less effective but they are in a minority.[22]

Moderately successful treatment of hay fever by the use of pollen orally has been reported by Iliff,[23] using a pre-seasonal method. However, adverse reactions, *e.g.*, diarrhœa, vomiting asthma or urticaria, occurred in 40% of cases. The parenteral method obviously gives a greater percentage of satisfactory relief, a smaller percentage of absolute failures and fewer unpredictable reactions.[24]

There is wide variation among allergists as to the maximum dosage to be given and as to the total dosage in pre-seasonal treatment. Brown,[25] Levin,[26] Waldbott[27] and Ellis[28] advised maximum doses varying, in each instance, from twelve thousand to a hundred thousand pollen units (1 pollen unit equals 0.64 protein nitrogen units). In determining efficacy of treatment, at any level of dosage, difficulties are encountered in establishing controls because of variation in air pollen concentration, presence of other sensitivities, inhalant or otherwise and the subjective method of assaying treatment. These have led many to believe that the optimal dose is one that must be established by trial and error in each case and may vary widely from one case to another. Rackemann[29] believes that pollen tolerance is more or less fixed for each individual and this can be determined by noting the level at which general reactions begin to occur. For him the optimal dosage is one that starts three or four doses below the level at which general reactions occurred in previous years. Hansel[30] endorses a very low final dose in preseasonal therapy, about one unit.

The occurrence of general reactions mars rapid and high dosage schedules. It makes high dosage difficult to achieve. Such reactions may occur at any time during the course of treatment. They begin with widespread dermal or respiratory symptoms; urticaria, pruritus, rhinitis, asthma. Headache or gastro-intestinal symptoms may occur. A shock-like state with loss of consciousness, fall in body temperature, etc., occurs occasionally. The cause of these reactions may be faulty selection of dose, or improper administration causing inadvertent intravenous injection, or, occasionally, the concurrence of a food allergy or the absorption from the air of sufficient inhalant antigen to which the patient is sensitive, to make, with the dose injected, a quantity too large for the patient's tolerance.

Some objective method of determining a patient's degree of sensitivity and therefore the amount of pollen therapy needed has always been an acute need. The discovery of the blocking antibody by Cooke and co-workers[31] in 1925 offered promise that some such yardstick had been found. It differs from the antibody "reagin", which is responsible for the skin reactions and for symptoms in susceptible persons, in its thermostability, its greater affinity for antigen, and its rapid disappearance from injected skin sites. In blocking the union of reagin and ragweed antigen, it is responsible for clinical relief in hyposensitized cases of hay fever. Increased levels of blocking antibody can be found not only in circulating fluids but in the tissues of treated hay fever cases.[32, 33, 34] Unfortunately, it has been found that no correspondence exists between the level of blocking antibody achieved and clinical improvement. One individual seems to need more antibody than another for relief of symptoms.

If skin and mucous membrane sensitivity to pollen varied directly with the degree of clinical sensitivity and with the amount of desensitization therapy given, these could be used as quantitative determinators in subsequent treatment. However, Baldwin and Glaser[35] found, in a series of treated cases, reduction in skin sensitivity in one-half the number. This half did not include all those helped by treatment and included some who derived no benefit. Reduction of mucous membrane sensitivity was found more regularly in adequately treated cases. Feinberg and associates[36] found a similar correlation but noted that, unfortunately, presence of infection in the upper respiratory tract and presence of other sensitivities in the subject, vitiated its reliability as a quantitative indicator of sensitivity.

The fact that neither blocking antibody level, reduction in skin sensitivity, nor, at present, mucous membrane sensitivity can be used as an indication, quantitatively, of clinical sensitivity to pollen, or level of desensitization, does not imply that one should not attempt to achieve a certain critical dosage level, as so many assume. The report of the study which follows will show

that only above a certain critical dosage level can one expect to achieve a favourable result in desensitization.

ANALYSIS OF CASE MATERIAL

The cases for study included a total of 78, 58 of which were treated in the out patient department of the Royal Victoria Hospital and 20 cases treated at the author's office in 1952. The age range of patients was six to sixty-six years; there was a slight preponderance of females in the group. Eight cases took perennial treatment; the remaining 70 took pre-seasonal treatment, beginning about March 20 or later. Twenty to twenty-five injections was considered the ideal pre-seasonal course, and was achieved by some, particularly cases treated privately.

Estimation of results based entirely upon a subjective estimation of symptoms by the patient is obviously a method into which inaccuracies will find their way. For this reason, physical examination where indicated and possible was done to confirm information given on questioning. The patients were classified as having no hay fever, or mild, moderate, or severe hay fever. Many of those with severe hay fever also had asthma due, presumably, solely to pollen sensitivity. In no case in this series were infective factors in the upper or lower respiratory tract thought to influence the clinical picture, and, in each case, where food or other inhalant factors existed, these were placed under concurrent control which was thought adequate.

The results as shown in Chart I clearly point to a correlation between low dosage and severity of symptoms, the severe hay fever symptoms all occurring in the cases which received less than 1,000 protein nitrogen units as a final dose. Among this dosage group are two cases free from hay fever and one case with mild hay fever so the correlation is not absolute, but it is nonetheless striking. Table I has been constructed using 1,000 protein nitrogen units as a critical level. It shows that among patients who received less than 1,000 protein nitrogen units as a final dose, 62% had severe symptoms and 32% had moderate symptoms, whereas among patients who received more than 1,000 protein nitrogen units as a final dose, 45% had no symptoms and 41% had mild symptoms. Grouping the severe and moderate cases together and the mild cases together with those with no hay fever, it is apparent that with final dosages under 1,000 protein nitrogen units, 94% of the patients did poorly,

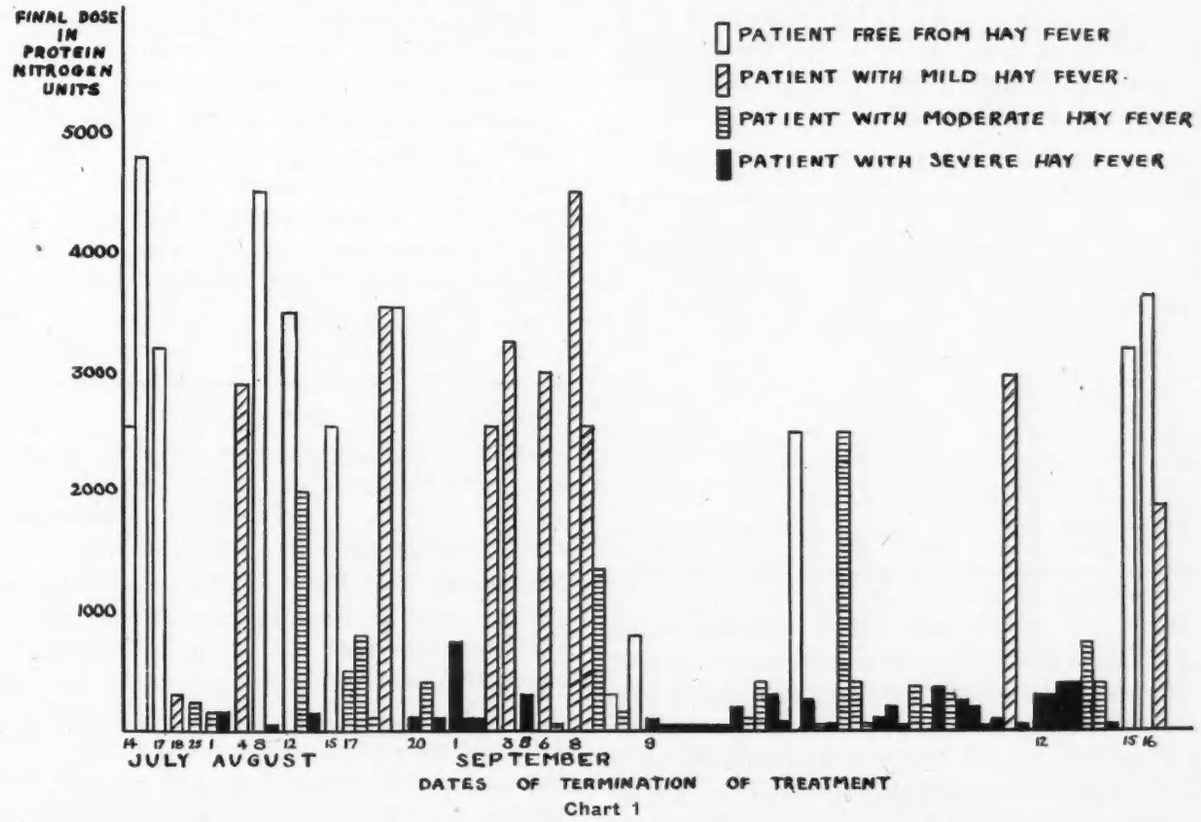

Chart 1

Canad. M. A. J.
June 1953, vol. 68

while with final dosages over 1,000 protein nitrogen units, 86% of the patients did well. Evidently, while some cases of ragweed pollinosis may achieve hyposensitization on small doses, the average case will require that a certain fairly high level of dosage be reached before good results will be obtained. Some exceptional cases will require very high dosage. In the field of immunization, an analogous occurrence is the achievement by most persons of immunity to diphtheria by injection of 2 c.c. of toxoid, while a few exceptional cases will require as little as 0.1 c.c. and some as much as 5 c.c.[28]

These results were obtained in a year of comparatively high ragweed pollen concentration in the Montreal area. Daily pollen counts from August 1 to September 9 totalled 1,777 in 1952 compared with 772 in 1951.

TABLE I.

	Number of cases	*Symptoms of hay fever*			
		None	*Mild*	*Moderate*	*Severe*
		%	%	%	%
Total cases unclassified as to dose.....	78	15	13	27	45
Cases receiving less than 1,000 protein nitrogen units as a final dose.....	56	4	2	32	62
Cases receiving more than 1,000 protein nitrogen units as a final dose.	22	45	41	14	0

Comparison of these results with those reported by other authors in previous years shows a close correlation of good results in adequately treated cases. These results have been achieved with widely varying top dosage levels, in most cases higher than ours. But in none, so far as can be determined, has there been an attempt to establish a critical level of dosage for attainment of good results.

The attainment of high dosage is militated against by irregular attendance at the clinic, by large local reactions at the injection site and by the occurrence of constitutional reactions. Irregular attendance, a greater problem in clinic than in private cases, not only leads to low dosage, but the irregularity itself tends to cause large local reactions and even constitutional reactions at relatively low dosages. Private patients have been said to do slightly better than publicly treated cases, probably due to the closer supervision and the effect that the generally more favourable life situation of the private patient has upon treatment.[22] In this series the factor of higher average top and total dosage would seem the more important reason for the better results obtained in private cases.

Elimination of excessive reactions, with unusually good results and attainment of high dosage by the simultaneous injection of antihistamine and antigen has been reported by Maietta[8] in 45 cases of ragweed hay fever. Eleven of our cases so treated but excluded from this series tended to confirm Maietta's results but the number of cases treated was too small to draw valid conclusions.

General or constitutional reactions are not infrequently due to injury to venules too small to permit detection of this by the usual method of withdrawing the plunger of the syringe. In such cases, intravenous injection of part of the material predisposes to general reactions. This can be obviated, in many instances, by the use of the finest bore needles (size 27) with the acutest bevels obtainable. The size 25 and 26 needles in frequent use tend to be more traumatizing, particularly when inserted through the skin with a quick thrust. Sharp, small bore, acutely bevelled needles, gently inserted as gauge 27 needles necessarily must be, penetrate the skin and subcutanous tissues with minimal venule damage. Another frequent cause of reactions is the use, at any time, of a dose too high for the individual's tolerance. It has been found expedient to so arrange the pre-seasonal schedule that injections with the strongest concentration of solution used should be near maximum dosage at a date no later than July 31. After this date, ragweed pollen begins to appear in the air, although only in minimal quantities until about August 15. High dosage given after July 31 is likely to be associated with constitutional reactions. When using solutions of 10,000 units per c.c., regardless of date, increments of increase greater than 500 units seem often to be associated with constitutional reactions and should be avoided.

SUMMARY

1. A survey of the present status of treatment of ragweed hay fever has been presented.

2. In a series of 78 cases of hay fever treated by specific desensitization, 95% of patients who for one reason or other received less than 1,000 protein nitrogen units as a maximum dose had

either severe or moderately severe symptoms, while 86% of patients who received more than 1,000 protein nitrogen units as a maximum dose were either symptom free or had mild symptoms only.

3. It is concluded that in the treatment of ragweed hay fever there is a critical level of dosage above which one may expect to get good results and below which results are almost uniformly poor.

4. Factors preventing achievement of high dosage; irregular attendance at the office or clinic, large reactions at the injection site or constitutional reactions, have been outlined and means of preventing these have been discussed.

My thanks are due to Miss Grace Allnutt, R.N., of the Allergy Clinic of the Royal Victoria Hospital and to Mrs. Anita Porter of the Outdoor Record Department of this hospital for valuable assistance rendered in the compilation of case material.

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THE MANAGEMENT OF MARIE-STRUMPELL SPONDYLITIS WITH SPECIAL REFERENCE TO LONG-TERM CORTISONE THERAPY*

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APPROPRIATE REST and adequate exercise remain the best form of treatment for Marie-Strumpell spondylitis. Before 1950, the only added treatment of proven value has been Roentgen therapy. This had failed to give as much or lasting relief as had been hoped. When cortisone appeared, its use was adopted as another adjunct to the basic program. Contrary to prevailing trends and for reasons to be given, study of a small number of patients was undertaken 2½ years ago, using cortisone intermittently rather than continuously. The results of this method have been satisfactory.

Selection of patients.—As a result of previous experience (in Shaughnessy Hospital), the impression was gained that most patients with rheumatoid disease tend to improve, or recover, if placed under ideal conditions for a sufficiently long period of time, even without specific treatment. Of more than 150 veterans of World War II with Marie-Strumpell spondylitis, some 35 had not, by 1950, achieved sufficient recovery to permit reasonably satisfactory wage-earning. Some of these 35 patients were badly disabled by deformities or by very severe persistent rheumatoid inflammatory activity and, with few exceptions, were not included. An economic factor, potential restorability to wage-earning, was the chief consideration in selecting this group of 11 male patients for long-term management with cortisone in order to test its practical value. Of the 11, four had been in hospital for some months before hormone therapy was started. The other seven patients, called in to hospital for treatment, were prevented from working full-

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Canad. M. A. J.
June 1953, vol. 68

time by active rheumatoid disease. It was felt that moderate subsidence of the inflammatory activity would permit all these patients to become economically self-sufficient. Although spondylitis was the primary disease in all, weight-bearing joints were involved in eight of the 11 patients and often shared equally with the back involvement in producing the total quantity of disability.

Plan of treatment.—All of these patients had previously had one or more similar hospital periods, without cortisone, without restoration of full wage-earning ability. The initial course of cortisone was given in the Shaughnessy Clinical Investigation Unit. Subsequent management was on an out-patient basis; at first at weekly intervals. Further cortisone was administered when necessary, usually without bringing the patient back in to Hospital.

(a) *Dosage.*—The standard dosage was 100 mgm. daily, given either intramuscularly or orally. Rarely was it necessary to increase, temporarily, the daily dosage to as high as 200 mgm. in order to obtain a satisfactory clinical response. Routinely, the supplementary courses ("booster") consisted of five or ten days of 100 mgm. daily given as one dose intramuscularly, or as 25 mgm. every six hours by mouth.

(b) *Duration of initial course.* — The initial course was intended to bring about maximal symptomatic remission. However, it was, nevertheless, terminated when, after substantial clinical gain, further improvement failed to occur on the maximum dosage tolerated well by the patient.

(c) *Indication for supplementary treatment.*— The short "booster" course was given whenever it seemed clinically necessary to maintain or retrieve the satisfactory remission. As a rule, increasing functional impairment rather than worsening of more objective criteria constituted the indication for re-treatment. We adopted this policy because most of the patients became fully employed and the primary objective of this project was to keep them at work. Each patient was re-treated according to the dictates of his own rheumatoid inflammatory process and no attempt was made to institute a uniform treatment-free interval between courses. Nor was the hæmatological picture reliable as an indication for re-treatment.

Results.—It is difficult to assess results by a rigid set of criteria in rheumatoid disease. This is especially true in spondylitis where spine and peripheral joints contribute variably in the disability. In these veteran patients, we have felt that the most important criterion was change in work capacity. Fig. 1 records these results. Degrees of work capacity were classified thus: Class 1—Complete work capacity with ability to carry on all usual duties without handicaps. Class 2—Work capacity adequate to conduct normal activities despite handicaps of discomfort or limited mobility of one or more joints. Class 3—Work capacity adequate to perform only little or none of the duties of usual occupation. Class 4—Largely or wholly incapacitated.

From the results (Fig. 1) it will be seen that four patients (D.M.L., C.W., T.W. and W.H.W.) showed marked improvement, an increase in work ability of two or more classes. Moderate

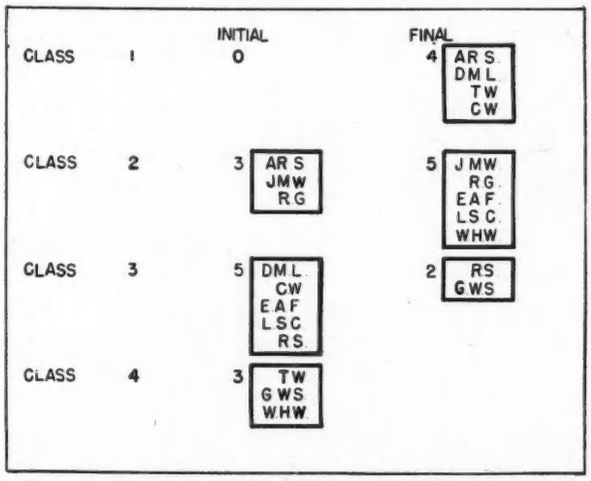

Fig. 1.—Results in the treatment of eleven patients with Marie-Strumpell spondylitis using intermittent cortisone, showing increase in work capacity.

improvement, an increase by one class, was shown by four patients (A.R.S., E.A.F., L.S.C. and G.W.S.) while three (J.M.W., R.G. and R.S.) showed little or no improvement.

Initially, four of the eleven were employed at part-time or light work, while at the present time ten are employed at full-time duties ranging from sedentary desk work to arduous manual labour.

Fig. 2 indicates the amount of cortisone given in the initial course and the length of time over which it was given. It shows the smaller "booster" courses, the route of administration and the interval between each. The duration of the period of observation and the total amount of cortisone given is listed at the left.

Change in physical examination.—Objectively none are worse. Those with marked increase in

work capacity show at least some measurable gain physically, with decreased inflammatory activity and improved range of movement.

Hæmatological effects. – During the longer initial course of cortisone the sedimentation rate tended to fall and to gradually rise again during the intervals between therapy. In only three cases (T.W., A.R.S. and E.A.F.) has the sedimentation rate remained consistently at a lower level than before starting therapy. The hæmoglobin and white blood count tended to rise during the initial course, only to fall gradually to the former levels. The eosinophil count showed no consistent variation related to cortisone treatment.

No change in the blood electrolytes or serum protein was discernible.

Route of administration.—The route of administration of cortisone did not appear to have any relationship to the results since the same results were noted with both intramuscular and oral cortisone in most of the patients. Intravenous and intra-auricular routes in the same dosage gave similar results.

Supplementary courses.—A rather surprising amount of symptomatic benefit was obtained from short, supplementary courses. So far as purely spondylitis symptoms were concerned, little difference between ½ gram in five days or one gram in ten days was noted. When active inflammation of weight-bearing joints was present, the ten-day booster course usually gave the better results.

Impairment of adrenal function —In the six patients whose urinary excretion of 17-ketosteroids was followed during the period of initial therapy, a change was observed consisting of a fall in the 17-ketosteroid level, which was more striking in those in whom the level was higher before treatment. This fall occurred in patients receiving both parenteral and oral cortisone.

The response to a single 20 mgm. intramuscular dose of ACTH (Thorne test) was established, before treatment, in a number of patients. Both eosinophil responses at four hours and 17-ketosteroid excretion over a six hour period after ACTH administration were measured. This information has been published[1] but, in summary,

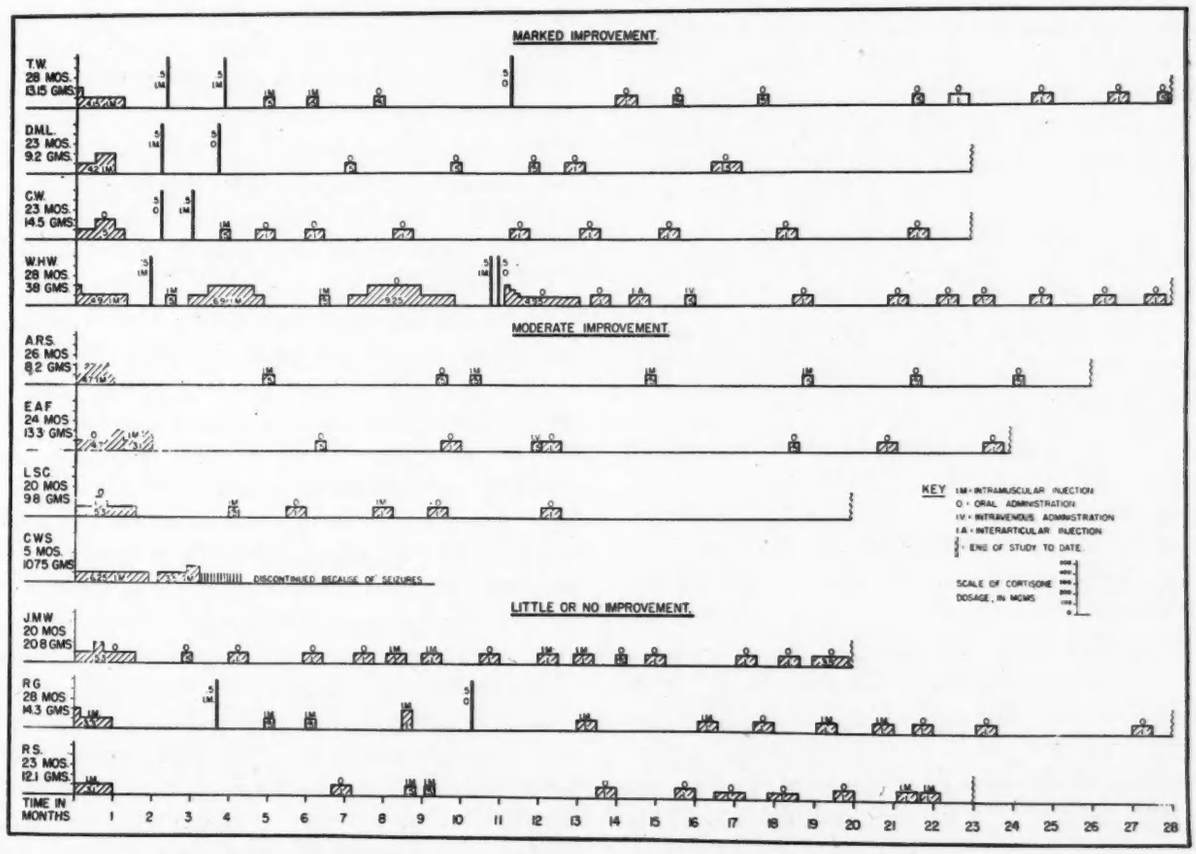

Fig. 2.—This figure records the total amount of cortisone used, the duration of the period of observation, the route of administration and the interval between courses.

it can be stated that a depressed response to ACTH for thirty days after the *initial* course parenterally, and twenty days when the oral route was used, was observed. Further studies are in progress to see whether the adrenocortical inhibition is correspondingly less with short courses of five and ten days.

Adverse effects. — These consisted of reactivation of well-controlled epilepsy during a long initial course in one patient, presumably due to hydration from salt and water retention.

While gastro-intestinal hæmorrhage occurred in two patients under treatment, there was no more reason to believe that this was due to the cortisone than to the aspirin compound consumed in the intervals between courses of hormone therapy. In one a new peptic ulcer was demonstrated by x-ray, while in the other, no such lesion could be found. Some retention of fluid occurred in two instances.

DISCUSSION

The intermittent rather than the continuous method of administration of cortisone was decided upon for the following reasons:

1. It was felt that the continuous use of an agent which resulted in the suppression of adrenocortical function might interfere with the spontaneous remission which, in the past, was encountered in a reasonable number of patients with rheumatoid disease.

2. To reduce the likelihood of the side-effects of continuous long-term administration. With the use of cortisone now on a wide scale, it has become apparent that a "tissue fastness" or resistance to cortisone develops in a good many patients in whom it is used continuously. This also we had hoped to avoid by using cortisone intermittently. We had also anticipated that following a "short burst" of cortisone, a return towards more optimal function of the adrenal cortex might occur.

After due consideration we adopted the change in work capacity or job performance as the main criterion for our evaluation of cortisone therapy in Marie-Strumpell spondylitis. The fact remains that all but one of these eleven patients are now working and this seems to us to be significant. Each patient had had previous admissions to hospital where basic, essential treatment had been employed, thus each had served as his own control.

The return of symptoms, with the cessation of cortisone, appeared to follow one of three courses: (1) Transient over-resurgence. (2) Rapid return of symptoms. (3) Slow return of symptoms. Over-resurgence was infrequent. Failure of cortisone, by this intermittent method of administration, would seem to be assured by a rapid return of symptoms. However, this was not invariably the case, as shown by the young man who had repeated, long courses over the first eighteen months, during which time he suffered a rapid return of symptoms on each occasion when cortisone was stopped. During the past ten months he has responded well to one gram supplementary courses with only a slow return of symptoms. Weight-bearing joints have been improved also by the concomitant use of compound F acetate* intra-articularly and he has now become self-supporting. If, after the initial course, the patient remains reasonably comfortable for three or four weeks without further cortisone, a good result may eventually be achieved.

As noted before, cortisone in these patients has been an adjunct to the basic program of treatment. Relief of pain made it much easier to provide emotional and physical rest. Exercise therapy was more successful. The physical gain obtained in the initial course was seldom lost when symptoms returned. Much of any such loss was quickly regained with the short supplementary courses. Cortisone proved to be a morale builder even in two of the three patients who were not materially benefited, for they could and do look forward to the comfort provided by the next brief course, though the effects were usually transient. The third patient was taken off cortisone because of persisting duodenal ulcer in the face of a poor response to treatment. Co-operation has been excellent and no one has asked that the treatment be stopped. These observations, together with the definitely improved work capacity of most of the group, influenced us in coming to the conclusion that there is merit in the intermittent technique of using cortisone at least in certain patients with Marie-Strumpell spondylitis.

The mechanism of action of cortisone in rheumatoid disease is still a matter of speculation. It is unlikely that the beneficial effect is related to the suppression of adrenal function. It would seem more reasonable to suppose that the

*Compound F was supplied by Merck & Co. Ltd., through the courtesy of Dr. J. H. Laurie.

activity of the rheumatoid process is dampened by cortisone which, perhaps, inhibits tissue response to the etiological agents. Following cessation of cortisone therapy, the disease process takes a variable length of time to be rekindled. The reason for this variation is unknown but may depend upon such factors as the intensity of the provoking stimuli and the rather nebulous quality known as host resistance. This concept seems to fit most of the clinically observed features. It has the advantage of recognizing the natural history of remission and exacerbation in the untreated condition and it served as a useful premise that resulted in our relatively satisfactory effort to employ cortisone intermittently at a time when its continuous use was universally advocated.

SUMMARY AND CONCLUSION

The intermittent use of cortisone in the treatment of a small number of patients with Marie-Strumpell spondylitis has proved an important adjunct to the basic program of treatment. Ten of eleven patients showed improved work capacity that can be attributed to the use of cortisone intermittently by short supplementary courses, over a period of twenty-four to thirty months.

The authors wish to thank Dr. M. M. Baird, Director of Medicine, Shaughnessy Hospital and the nursing and technical staff of the Clinical Investigation Unit for their assistance throughout this investigation.

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CASE REPORTS

PURPURIC ERUPTION DUE TO "SEDICIN"

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IN AUGUST 1952, Dr. J. R. E. Morgan of Peterborough referred to me two patients with bizarre, widespread purpuric eruptions. These eruptions in each case started on the feet and legs, spread to the thighs and later to the buttocks, lower trunk and forearms being most pronounced below the knees. The eruption resembled that described many years ago by Schamberg[1] as a peculiar, progressive, pigmentary dermatosis. In each case, however, the eruption had spread much more rapidly than in typical cases of Schamberg's disease. Morgan discovered that each patient had taken Sedicin prior to and during the course of the eruption. Following withdrawal of the drug, the skin gradually returned to normal.

Sedicin is a patent medicine sold by druggists without prescription and advertised widely in the daily press as a "safe remedy for insomnia and to relieve nervousness". It is acetyl-diethyl-bromo-acetyl urea and is an open chain ureide closely related to Sedormid and to the barbiturates. Joron, Downing and Bensley[2] report two deaths from Sedormid, in both of which there was marked central nervous system depression prior to death. They also report three other fatal cases in two of which there was cerebral purpura. The statement is made that Sedormid produces purpura even in small doses but there is no clear evidence that related ureides cause it.

The following case is briefly reported, together with a photograph of her feet and ankles, to show the type of eruption present.

A white female patient, aged 62, was first seen September 29, 1952. An eruption had started on her feet in July. This had spread to her legs, thighs, abdomen, buttocks and finally to her arms. There was mild pruritus. Because of the two previous patients who had eruptions produced by Sedicin, this patient was carefully questioned regarding the taking of medicine. She purchased Sedicin without prescription and took one tablet each night for one week in July, then increased to two tablets each night because she did not sleep well, and finally to three tablets. Following three tablets at bedtime she stated that she "felt stupid" the next day. She had taken three boxes of Sedicin prior to being seen.

The eruption developed after she started taking the Sedicin and became rapidly worse. The patient had applied various topical remedies to the skin without relief and it was becoming so pronounced and extensive that she was alarmed about it. On examination she appeared to be in excellent general health.

Involving the areas of skin mentioned previously there was a brownish red eruption, most pronounced on feet, ankles and knees and least pronounced on arms. The eruption consisted of patches of discrete and confluent, small macules which had a slight scale and in which there were minute hæmorrhages appearing like dots of cayenne pepper. There was also some brownish pigmentation about the small macules. The mouth and throat appeared normal. Capillary resistance test was normal.

Laboratory investigation.—Urinalysis, normal. Leucocyte count, 9,600. Bleeding time, 1 minute 35 seconds (normal). Coagulation time, 5 minutes 40 seconds (normal). Differential leucocyte count: neutrophiles 61.6%; eosinophiles 1.8%; lymphocytes 30.2%; endothelials 6.4%.

The patient was immediately advised to discontinue the use of Sedicin and all similar or related hypnotics and sedatives. The eruption began to fade within a few days

Canad. M. A. J.
June 1953, vol. 68

and had almost completely faded by the end of three weeks although slight pigmentation of feet, ankles and legs persisted for three months.

SUMMARY

Three patients with purpuric eruptions produced by Sedicin are reported.

It is probable that there are many similar cases which remain unrecognized.

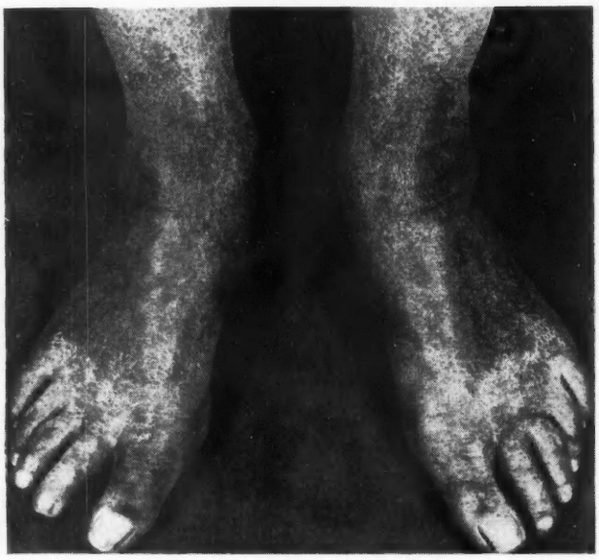

Fig. 1

In view of these toxic reactions, it is strongly recommended that the Department of National Health and Welfare forbid the indiscriminate sale to the public of Sedicin, Sedormid and other identical or related open chain ureides.

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HERNIA COMPLICATING PREGNANCY

S. C. ROBINSON, M.D., *New Denver, B.C.*

A 25 YEAR OLD white woman was seen at intervals during her fourth pregnancy. She was in good health, and her previous obstetrical history was normal. Prior to her first pregnancy, an appendectomy had been performed, and also a "lump" had been removed. No records had been left by the surgeon, and she was unable to be more explicit.

Physical examination showed her to be a multiparous woman, pregnant but well. The only abnormal finding was a mitral systolic murmur with no cardiac decompensation. There was no evidence of hernia. On August 15, 1952, about a month before term, she noticed a slightly painful mass in the right groin. This would subside when she was recumbent. Examination at her next visit showed a right indirect inguinal hernia, which was reducible. She stated on further questioning that she had had a rupture as a child which had cleared spontaneously and that again in 1949, she had noticed a slightly tender swelling in the right groin after lifting a child. It had always subsided after rest and in a year had stopped appearing.

Two weeks antepartum the swelling failed to subside at rest and became more tender, but not really painful. She was told to avoid all straining and on September 20 the membranes ruptured spontaneously. After a 2 hour labour, she was delivered of a normal infant. Ergonovine was used intravenously after the second stage, but the placenta did not seperate. After an hour it was manually removed. The puerperium was uneventful with the hernia unchanged, and giving no distress. She had bathroom privileges from the second day.

On the 5th day the hernia became larger and more painful. She was kept in bed with the foot elevated and ice applied to the groin. Although no signs of obstruction developed, there was no improvement, and the following day an operation was carried out under spinal anæsthesia. A tight adherent mass the size of a pullet's egg was found, protruding through the external inguinal ring. It appeared to be cystic. Comparatively dense adhesions were broken down by blunt dissection and the canal was opened. The sac contained a firm œdematous structure about 1.5 cm. in diameter which ran from the mass through the canal, internal ring and on to the side of the partially involuted uterine fundus. In the abdomen, it appeared to be the upper edge of the broad ligament. It was divided as close as possible to the fundus, and the repair completed without difficulty.

The pathological report stated that the specimen was "hernial sac containing parovarian cyst and portion of fallopian tube; no actual ovarian tissue present in the sections examined".

Fortunately there were no difficulties with either labour or operation in this case. It does,

however, raise the question of when to temporize and when to operate when inguinal hernia is a complication of late pregnancy. A strangulated hernia of bowel might present a far more serious problem than was found in this unusual case. Should it occur in conjunction with labour it might well be disastrous.

The pathological examination of the specimen was made by Dr. H. H. Pitts, of St. Paul's Hospital, Vancouver, B.C. I am indebted to him for this report.

ANAPHYLACTIC SHOCK FOLLOWING PROCAINE PENICILLIN INJECTION*

J. MIGNAULT, M.D. and
H. S. MITCHELL, M.D., *Montreal*

WITH THE PUBLICATION of 6 cases treated with penicillin from February to August 1941,[1] a new drug appeared which brought some hope in the treatment of infection. Further trials showed the high value of the new drug. But, later on, some shadows appeared in the picture and the miraculous antibiotic was found to be capable of harming the one it was supposed to help. Reactions of different types and gravity were observed: local irritation, urticaria, angioneurotic œdema, erythema multiforme, serum-sickness, mental disorder and, rarely, anaphylactic shock.[2, 4] Duemling reported an incidence of reactions of 10%, including Herxheimer's reactions, in 17,879 patients.[5]

The first case of anaphylactic death from penicillin was reported by Waldbott[6] in 1949. A 39-year-old female with a history of asthma had received three courses of penicillin in the past. Following the third one, she developed severe urticaria, aggravation of her asthma, joint pains and a slight fever about one week after the administration of penicillin. Suffering again an aggravation of her chest condition, she reported to her doctor who recommended crystalline penicillin. Soon after the injection, she felt a strange taste in her mouth and experienced a feeling of swelling and tightness of her throat and nose. Her face became flushed. She became extremely cyanotic and felt itchy "all over". While leaning over the kitchen table and asking for a glass of water, she collapsed and died.

About one year later, Burleson[7] reported a second case. A 40-year-old male had had, in the past, 3 injections of penicillin. 15 minutes after the intramuscular injection of 200,000 units of crystalline penicillin, he went into shock. This patient recovered after the intravenous injection of 30 to 40 mgm. of diphenhydramine hydrochloride.

More recently, other similar reactions have been observed. In January 1952, Yuval[8] reported the case of a 53-year-old mechanic who, after he was given 300,000 units of procaine penicillin intramuscularly, immediately felt dizzy and was forced to lie down. His respiration became stertorous and he could not respond to questioning. His radial pulse and apical beat were impalpable. No heart sounds could be heard. After 2½ minutes, his heart beat returned and he recovered completely, without any treatment, one hour later.

Thomson[9] reported the case of a 67-year-old male admitted to hospital to have a bougie passed for a urethral stricture. He had had one reaction before with procaine-penicillin. As this first reaction was thought to be due to procaine, it was decided to use crystalline penicillin. Following the intramuscular injection of 300,000 units, he developed nausea, started to retch and produced a little amount of vomitus. He became increasingly cyanotic with shallow gasping respirations and entered into a stage of collapse. His pulse could not be felt. Although he was given adrenalin and nikethamide he died.

At a meeting of the New York Allergy Society, Siegal and Shepard[10] reported three cases of anaphylactic shock following penicillin injections. The rest of the 60 members had personal knowledge of 3 other cases not reported.

Harpman[11] reported the case of a 3½-year-old child who died 3½ hours after the intramuscular injection of procaine penicillin. He had had one injection the day before without any reaction. One hour after the second one, he lost consciousness, became dyspnœic and died.

Higgins and Rothchild[12] reported the case of a 57-year-old labourer admitted to the hospital on April 1952 for the removal of a nodule of the right breast. 300,000 units of penicillin were injected intramuscularly, after withdrawal of the syringe plunger, ensuring that the needle was not in a vein. A minute and a half later, the pa-

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Canad. M. A. J.
June 1953, vol. 68

tient suddenly became cyanotic and coughed. A tonic muscular spasm developed. No blood pressure or pulse could be obtained. He died 10 minutes after the injection. In his past history, he had had a local anæsthetic with procaine in 1949 and had received procaine penicillin in 1950 without any reaction. No family or personal history of any kind of allergic phenomena was elicited.

Mr. C.L., a 58-year-old white male gave a history of chronic bronchitis of 5 years' duration. He was given penicillin on 5 occasions for this condition without any reaction. His condition became worse in the fall of 1951 and he reported to this hospital for investigation in February 1952. A diagnosis of bronchiectasis of the right lower and middle lobes was confirmed by bronchogram.

Because of his age and the condition of the remainder of his lungs, it was decided to treat him conservatively. He came back periodically to the out-patient department for follow-up and medication.

On July 11, 1952, he came to the clinic, complaining of increased cough, shortness of breath, chest pains, abundant muco-purulent sputum and loss of strength. On physical examination, both lungs were resonant and the expiration was prolonged. Coarsely vesicular râles were noticed at the right base anteriorly and posteriorly. Rhonchi were observed all through both lung fields. It was decided to give the patient 600,000 units of procaine penicillin daily for three days.

The first injection was given from a 5 c.c. vial of procaine penicillin, containing 300,000 units per c.c. at about 12.00 noon, on that day. The rest of the vial was used on other patients who did not show any reaction.

As the patient was leaving the hospital, approximately 5 minutes after the injection, he experienced swelling and itchiness of his hands and fingers. Rapidly, this sensation extended to his face and he felt "bad all over". His chest became "tight".

He came back to report the abnormal sensation and was told to lie down. A doctor was called and he found the patient lying in bed, unconscious, without palpable radial pulse or audible blood pressure sounds. He was somewhat cyanotic and his respirations were shallow. He was taken to the ward where adrenalin was given subcutaneously and the foot of the bed elevated. This medication was repeated 15 minutes later and about 10 to 15 minutes after the second injection, the pulse could be felt and his blood pressure was 70/30. The patient started to talk but was still very confused. He was then given 50 mgm. of diphen-hydramine hydrochloride orally every 6 hours for two days. His blood pressure returned to its normal level of 110/70 in the next 12 hours.

The next morning, the patient could describe the early sensations he had experienced immediately after the injection of procaine penicillin; remembered being told to lie down but did not recall anything from then until the time he became conscious during the night. He remained unconscious for about 12 hours.

To determine whether the reaction was due to penicillin or to procaine, it was decided, two weeks later, to perform intracutaneous injections of diluted solutions of procaine, penicillin and procaine penicillin. The solutions used were: (1) procaine 0.1%; (2) crystalline penicillin, 3,000 units per c.c.; (3) procaine penicillin, 3,000 units per c.c. All dilutions were prepared with saline. 1/20 c.c. of each solution was injected as follows: (1) procaine solution and crystalline penicillin into separate sites in the right forearm; (2) procaine penicillin into the left. The patient showed no reaction to procaine or crystalline penicillin but to procaine penicillin he showed an extensive local reaction with pseudopodia and some of the early symptoms of the previous reaction, *i.e.*, itchiness, sensation of swelling of the arm, etc. To prevent a more serious reaction, a tourniquet was applied and adrenalin given. Attempts to show the presence of antibodies by the Prausnitz-Küstner technique were unsuccessful.

DISCUSSION

If one considers the amount of penicillin injected daily throughout the world, the number of the severe reactions described above is very small. Nevertheless, it seems worthwhile to bring them to the attention of the medical profession because they can be prevented in some cases.

Lepper *et al.*[13] and Mark, Lepper, Dowling *et al.*[14] have demonstrated a higher incidence of reactions with penicillin in oil and beeswax than with crystalline aqueous penicillin of procaine penicillin in oil. These authors claim that the preparation in oil and beeswax is confined to one site for a longer period and, because of the local irritation, there are probably present many damaged proteins which may be conjugated with penicillin and render it antigenic. Waldo[15] demonstrated in rabbits that penicillin must be bound to human albumen to become antigenic.

From the higher incidence of more severe reactions in the last two years, one may postulate that the sensitizing properties of penicillin are increasing with the widespread use of the drug. This is in agreement with Risman's[16] findings.

There is also some evidence that people with a past history of penicillin reaction or other types of allergic phenomena are more likely to develop penicillin reactions of greater severity than individuals with no such history. Trichophyton infections or any fungus infection in the organism may predispose to penicillin reactions. Sanchez-Cuenca[17] desensitized a patient who had developed a giant urticaria after penicillin by using increasing doses of penicillium extracts. He also studied the stools of eight persons, two of whom were sensitive to penicillin. He found penicillium to be present in the stools of the two sensitive persons and in one who was not sensitive. Ris-

man[16] demonstrated a higher incidence of positive skin tests in people with a history of trichophyton infection. It is possible that both penicillin and trichophyton have a common nucleus responsible for cross-sensitization.

CONCLUSIONS

From these facts, it is evident that one must be careful in the use of penicillin therapy. Also, one must condemn the dispensing of penicillin tablets, lozenges, chewing gum, tooth paste and candy over the drug store counter for sore throat or common cold. When penicillin therapy is indicated, the physician should never fail to ask the patient these questions: has he ever received penicillin in the past? Has he ever had reactions, even mild ones, to the drug in the past? Has he had any fungus infections? One should give penicillin no longer than is clearly indicated. If a mild reaction occurs, the nature of which might possibly suggest sensitivity to penicillin, one should pause and consider before continuing therapy.

SUMMARY

1. The literature on anaphylactic shock following penicillin injections has been reviewed.

2. One case is presented who survived. The antigen appeared to be the procaine-penicillin compound.

3. Predisposing factors to penicillin reactions are discussed.

4. The need for careful evaluation of any patient prior to the administration of penicillin is stressed.

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ACTINOMYCOSIS OF FOREARM

P. G. FINCH, M.D., *Kimberley, B.C.*

THIS MAN was first seen by me in March, 1952 with the following history:

He came to Canada from the Treviso province of Northern Italy a year previously. He had noticed a vague swelling in the upper part of the right forearm before he left Italy. During the next year the forearm gradually became painful, the swelling increased, and a second swelling appeared below the first.

When I saw him the upper swelling appeared to be attached to the flexor belly of the muscles, and about 2" in diameter. The lower swelling was a little smaller though more prominent, and deep fluctuation could be detected. The skin moved freely over both areas and was not thickened or reddened. X-ray was negative.

He was operated on by Dr. O'Callaghan ten days later, and both masses were dissected free from the flexor muscles and tendons. The lower mass was cystic, and contained yellowish milky fluid; no "sulphur granules" were noted.

Pathology report was as follows:

> A granulomatous type of reaction composed of tubercle-like formations containing sulphur granules characteristic of actinomycosis or ray fungus. These will be identified by the use of special stains but there is very little doubt of their nature in the H and E sections. The granulomatous reaction is rather heavy and occasional foreign body giant cells identified. There is marked peripheral fibrosis. Portions of the granuloma appear to be present in the small amount of striated muscle tissue which is found in most of the sections.
> Diagnosis: Actinomycosis of forearm.

He was given 800,000 units of penicillin daily for seven doses in the postoperative period, and when a small collection of fluid formed at the lower end of the incision this was aspirated (culture negative) and replaced by a solution of crystalline penicillin. The fluid did not reform, and the wound healed firmly by first intention, and the man was back at work within three weeks.

A month after operation he was started on a six weeks' course of penicillin 1.2 million units daily, and this we believe will complete the cure.

This patient appeared to be in excellent health apart from this focus of infection. Chest x-ray was negative. Barium x-ray showed a normal cæcum. He had a small discharging sinus in his upper jaw which was negative for fungus on culture.

Canad. M. A. J.
June 1953, vol. 68

It would appear that this must have been either a blood-borne infection from some other focus (and no other focus was substantiated in this case); or it must have been from a puncture wound. He can remember no injury to this forearm except a wooden splinter wound when he was about seven or eight years of age.

TREATMENT OF CARDIOGENIC SHOCK BY PROLONGED ADMINISTRATION OF NOR-EPINEPHRINE*

A. ENGLISH, M.D., *Montreal*

THE IMPORTANCE of profound hypotension complicating myocardial infarction cannot be stressed too strongly when its associated high mortality rate (80 to 90%) and its relative frequency (10%) are noted.

It has been suggested that since hypotension lessens the work of the damaged myocardium, it is a useful compensatory mechanism. However, as Hellerstein *et al.*[4] point out, a distinction must be made between mild transitory hypotension and the more severe prolonged hypotensive states. The experimental work of Corday *et al.*[7] indicates that a fall in blood pressure, by reducing coronary artery blood flow, may cause extension of the original infarct and may even, in the presence of narrow coronary arteries, produce new areas of infarction.

Although cardiogenic shock is undoubtedly related to abrupt destruction of a portion of the myocardium, its pathogenesis remains obscure.[8, 9, 10] The degree of hypotension cannot always be correlated with the size of the infarct.[8, 10] However, at post mortem when an infarct is found to be extensive, there can be no certainty that its size is not, to an extent, a consequence of the previous hypotension.

In the past, attempts to re-establish adequate blood pressure levels have led to the use of such pressor amines as epinephrine, ephedrine, Amphetamine, Neo-Synephrine and Desoxyphedrine. All have undesirable side effects, the most important of which is a tendency to produce ventricular irritability.[11] Moreover, the general experience has been that these drugs are without any effect, or are only transiently beneficial, in combating cardiogenic shock.[12] Interest in the treatment of this important complication consequently lagged.

Within more recent years, the very encouraging results obtained from the use of intra-arterial blood and plasma,[13, 14] and more particularly from the newer, less toxic pressor amines, nor-epinephrine[2, 3] and mephenteramine,[1, 4] have revived interest in the treatment of this almost universally fatal complication.

The following case is presented in the hope that it may enhance this renewed interest, and because it is perhaps unique in that the patient's blood pressure was maintained at satisfactory levels by the continuous use of nor-epinephrine for a period (12 days) longer than has yet been reported.[2, 3]

Mr. J.R.A., a 62-year old white male, was admitted to the Queen Mary Veterans' Hospital in the early morning of October 27, 1952, complaining of severe constricting retrosternal chest pain and marked shortness of breath of approximately 4 hours' duration.

The patient was well-known at the hospital. During the past six years he had been admitted on seven occasions for treatment of long-standing chronic bronchitis, asthma and emphysema. In 1947 the right upper lung lobe was removed because of massive bleeding from a non-specific chronic abscess in that region. One month before his present admission his blood pressure was recorded at 140/90 and an ECG was normal except for prominent P waves in Leads II and III.

On admission the patient was fully conscious, but extremely apprehensive, dyspnœic, and cyanotic. The skin was cold and clammy. His temperature was 97° F. by axilla, the carotid pulse rate 80 per min., and the respiratory rate 45 per min. The systolic blood pressure was recorded at 70 mm. Hg. Later no blood pressure reading could be obtained by either auscultation or palpation. The heart sounds were not heard, largely because of coarse rhonchi throughout both lung fields. The ECG was characteristic of acute posterior myocardial infarction.

The patient was placed in an oxygen tent, and anticoagulants and prophylactic antibiotic therapy were started. He was also given 10 mgm. of Neo-Synephrine every 15 minutes, at first intravenously and later subcutaneously. By 9 a.m. on October 27 he had received a total of 200 mgm. of Neo-Synephrine without any obvious effect on the blood pressure (see Fig. 1). At 10 a.m. a solution of nor-epinephrine (4 mgm. per 1,000 c.c.) was started by continuous intravenous drip at a rate of about 30 drops per min. (0.11 μgm. per kilo. per min.). Almost immediately the radial pulse became perceptible. As blood pressure readings by auscultation were still unobtainable it was decided to record those made by the palpatory method. At 1.30 p.m. the systolic blood pressure was 100 mm. Hg. At 2.30 p.m. the supply of nor-epinephrine became exhausted and, in spite of the use of another pressor substance, Vasoxyl (methoxamine hydrochloride), his blood pressure again began to fall. It rose later when nor-epinephrine therapy was reinstituted.

During the following 12 days, a continuous intravenous drip of nor-epinephrine was administered. As noted in Fig. 1, whenever the rate of drip was reduced purposely or inadvertently, the blood pressure promptly fell to shock levels. The patient remained anuric for the first 36 hours of his hospital stay, the blood urea nitrogen

*From the Medical Service, Queen Mary Veterans' Hospital, Montreal.

rising steadily and reaching a maximum of 42 mgm. % by October 28. When normotensive blood pressure levels were reached and maintained, the urinary output increased and the blood urea nitrogen fell. On November 2nd Vasoxyl was discontinued abruptly without ill effect, the patient having received a total of 600 mgm. of this drug.

At 5 p.m. on November 6, following a high saphenous cut-down, the patient complained of chills, and the temperature rose to 104° F. A blood culture taken at the time was positive for Staphylococcus pyogenes, and antibiotic therapy was consequently intensified. For three days the patient ran a remittent temperature which gradually returned to normal by November 9.

Until now the patient had remained fully conscious. His morale, however, was poor and his appetite and general condition were deteriorating. Pain was continually present at the sites of three previous cut-down incisions, two of which were suppurating, and along the course of thrombosed superficial veins in the lower limbs. Furthermore, increasing technical difficulty was being experienced in maintaining the intravenous drip. It was now becoming evident that the patient's blood pressure would probably not be maintained if the nor-epinephrine were to be discontinued. It was decided therefore to try the effect of adrenal cortical hormones, which have a known hypertensive action presumably through potentiation of endogenous nor-epinephrine.[5] Accordingly at 4.30 p.m. on November 8 nor-epinephrine was discontinued (12 days after starting this therapy). At the same time 25 c.c. (750 units) of the rapidly acting aqueous adrenal cortical extract were administered in 500 c.c. of fluid as an intravenous drip over a six hour period. This was immediately preceded by, and also followed by, 25 c.c. of the same substance injected intra-muscularly. The patient was also started on cortisone,* 100 mgm. daily orally in divided doses; later the dose was reduced to 75 and then to 50 mgm. daily. For the first time since admission the patient's blood pressure did not drop when nor-epinephrine was stopped. Gradually the patient became more cheerful and his appetite improved remarkably. There was no evidence of fluid retention, serum electrolyte levels remained within normal limits and blood pressure readings could be determined by auscultation. For eight days nor-epinephrine therapy was discontinued, except for a short period on November 10 when a slight drop in blood pressure occurred.

On the morning of November 16, 50 mgm. of Demerol were given for limb pain. His blood pressure promptly fell to hypotensive levels, but was quickly restored again by nor-epinephrine.

On the afternoon of November 17 blood pressure again fell and from then until he died it was necessary to administer nor-epinephrine almost continuously. An ECG on November 18 showed changes consistent with extension of the myocardial damage. His condition gradually deteriorated and congestive failure appeared. Death occurred on the afternoon of November 19.

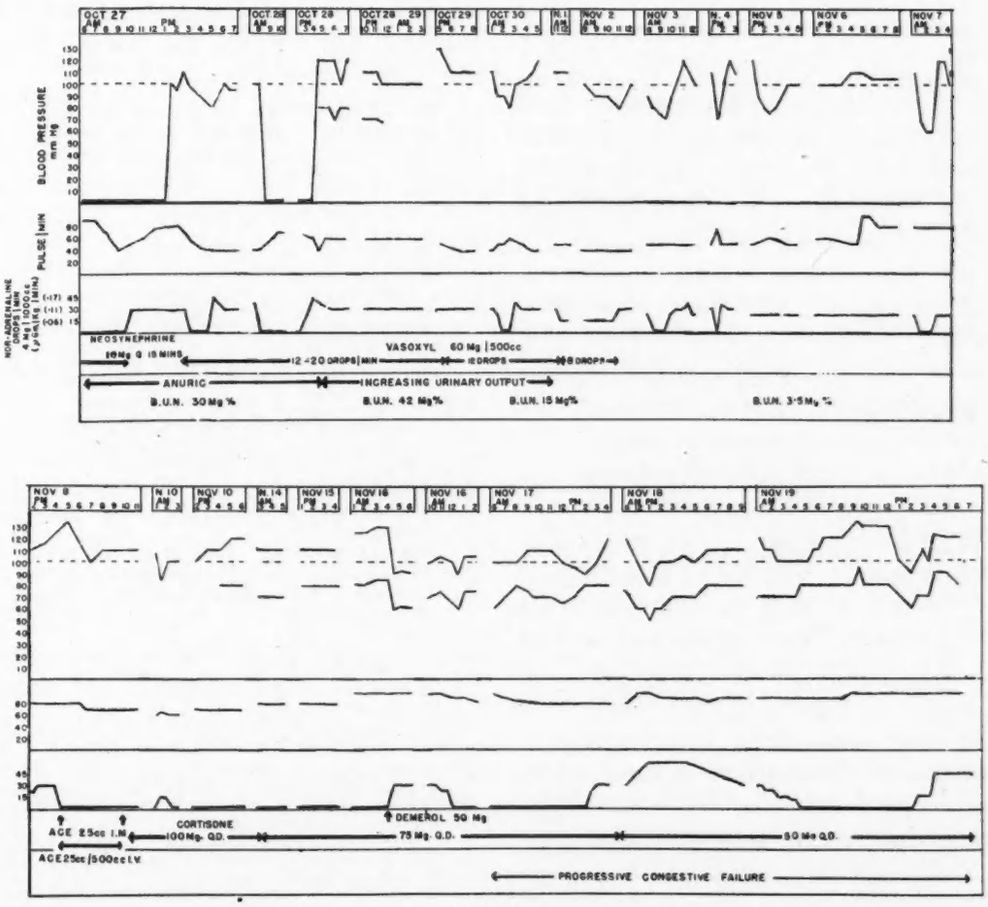

Fig. 1

During his hospital stay, he received a total of 148 mgm. of nor-epinephrine.

Autopsy findings.—(Permission was obtained for examination of the heart only.) All coronary arteries were markedly sclerosed and tortuous. The lumen of the circumflex branch of the left coronary artery was patent, but narrowed. This narrowing was much more pronounced throughout the anterior descending branch of

*Johnson *et al.*[6] have demonstrated in dogs, that the size of the infarcts following occlusion of coronary arteries were much less extensive in those animals treated with cortisone than in the controls.

the left coronary and at a point close to its origin a lumen could not be defined grossly. 3.5 cm. from the origin of the right coronary artery, a reddish-grey thrombus was found totally occluding the lumen.

The heart was large and weighed 450 gm. In the posterior wall of the right ventricle, a remarkably soft, pale area was found measuring 2 x 3 cm. This area was thinner than normal and at its central portion measured 1 mm. in cross section. Similar, but not as marked changes were found in the posterior portion of the interventricular septum.

Microscopically, sections of the arteries showed typical advanced atherosclerotic changes and an organizing thrombus occluding the right coronary artery. Sections taken from the above-noted, soft, pale area showed changes consistent with a three week old infarct. Similar findings were noted in sections from the posterior aspect of the interventricular septum.

DISCUSSION

This case demonstrates well the potent pressor action of and notable lack of tachyphylaxis to nor-epinephrine. No evidence of ventricular irritability, of central nervous system stimulation or of increase in general metabolism was observed clinically.

Although the patient's life was undoubtedly prolonged, this case by itself cannot be considered to represent any real therapeutic triumph. One might rightfully therefore question the wisdom of attempts to re-establish normotension in such individuals. Amongst the patients who die following the onset of hypotension, there is undoubtedly a large group where the cardiac damage is so extensive that there is inadequate functioning myocardium to carry on properly the work of the heart. The post mortem findings suggest this explanation in the above case. On the other hand, in slightly or moderately damaged hearts, the maintenance of an adequate blood pressure, essential to good coronary artery blood flow, can prevent further irreversible damage. It is in this latter group that treatment designed to restore adequate blood pressure levels may lead to recovery. Since there is no definite way of determining the outcome, treatment of the individual case is justified. Spontaneous recovery can occur, however. This is more likely to happen when the blood pressure does not fall below a systolic of 90 mm. Hg. (100 mm. Hg. in a previously hypertensive patient).[15] Therapy designed to elevate the blood pressure in such an individual is probably best deferred. However, when signs of shock appear and/or the blood pressure is unusually low, the sooner adequate blood pressure levels are re-established, the better may be the eventual result. Obviously, when delay occurs the opportunity for progressive possibly irreversible myocardial damage is enhanced.

SUMMARY

A case of myocardial infarction complicated by shock is presented. The patient's blood pressure was maintained at adequate levels principally by the use of nor-epinephrine, continuously for the first 12 days of his illness, and intermittently for the final 11 days.

The importance of prompt treatment of the severe hypotensive state is stressed.

Older and newer therapeutic attempts to achieve this purpose are briefly noted.

The author wishes to express his appreciation to Dr. J. S. Palmer, Consultant in Cardiology, Queen Mary Veterans' Hospital, for his assistance in preparing this report.

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In checking on results of the treatment of alcoholism, many investigators use continuous abstinence for a given period of time as the measure of success, and consider relapses as definite signs of failure. According to some therapists, however, a relapse is not necessarily a sign of failure but is even a desirable event in the course of treatment. Thus, J. A. Hobson (London) points out how difficult it is to convince an alcoholic that he will never be able to become a controlled drinker and that he must remain a total abstainer the rest of his life. The patient cannot bring himself to believe this truth absolutely until he has relapsed at least once. Frequently, after he has been abstinent for 6 months or more, is feeling well and has no desire for alcohol, he becomes confident that he can take one drink without upsetting his sober behaviour. He experiments, and the end results are always the same: the alcoholic who tries to drink sooner or later gets drunk.—*Alcoholism Research*, April, 1953.

SPECIAL ARTICLE

THE SERVICE BENEVOLENT FUNDS

A. D. KELLY, M.B., *Toronto*

FROM TIME to time the Executive Committee and the Committee on Economics have examined the operations of the Benevolent Funds, usually in response to a protest from a doctor that his accounts were being arbitrarily and drastically reduced. In the process of investigation I have learned something of the function of these Funds and have encountered misconceptions which are sufficiently widespread to justify this attempt at clarification.

In the first place these Funds are not Government funds and they are not administered by Government departments. At the end of World War II each of the three branches of Canada's armed forces had accumulated sizeable amounts of money in the form of non-public funds mainly consisting of canteen profits. It was logically recognized that this money belonged to the troops and it was decided to disburse it for the benefit of ex-servicemen who needed help. Accordingly the Army Benevolent Fund was set up under a Federal Act while the money belonging to Naval and Air Force personnel was turned over to the Canadian Naval Service Benevolent Trust Fund and the R.C.A.F. Benevolent Fund, both of which were already in existence, being supported through voluntary donations and contributions from canteens and messes.

The governing body of each consists of public spirited citizens who serve without remuneration and they have established voluntary committees in cities and towns and at service establishments where required. A small head office staff of full-time paid officials and, in some instances, full-time counsellors in the provinces are maintained.

Taking the Army Benevolent Fund as representative of the others, a few figures may be of interest. A capital sum of $9,000,000 was originally made available and it is planned to disburse this over a period of thirty years. Some of the capital and all of the interest is utilized each year. An annual budget of $470,000.00 is typical; $70,000.00 goes for administration and counselling services and $400,000.00 to veterans who need help. About $250,000.00 of this is expended in payments to doctors and hospitals, the remainder going to pay outstanding debts to finance companies, tradesmen and others. The Army Fund receives through its provincial committees about 6,000 applications for assistance per year and of these, 2,500 are approved and acted upon. The limitations of the available funds make it necessary to decline certain justifiable requests for help and to utilize what is available for as many as possible.

To be currently eligible for assistance, an army veteran must have served in World War II. It is anticipated, however, that after April 1953 contributions to the Funds will be received from the canteen profits of Canada's active forces, thus rendering all servicemen and their dependents eligible for help.

It should be emphasized that no eligible person is entitled as of right, to financial assistance. All payments made out of the funds are ex-gratia payments and they are authorized only after thorough investigation of need. Friendly counselling service is offered to applicants who find themselves in financial difficulties and debt repayment plans are frequently advised. These plans often permit the patient to pay the doctor in full in monthly instalments, particularly where the Fund is paying some of the other creditors. Also, through the counselling service applicants are strongly urged to subscribe to a plan of medical insurance and in fact this is sometimes a condition which the Fund will insist upon before awarding financial help.

Although the Funds operate along generally similar lines, some difference in practice is observed. For example, the R.C.N. and R.C.A.F. Benevolent Funds may utilize loans to beneficiaries to help them solve their problems, while the Army Fund makes only cash grants to settle the affairs of the screened applicants.

In respect of medical accounts, difficulties and misunderstandings arise most frequently under the following circumstances. A veteran or his dependent consults the doctor for illness and treatment is undertaken and brought to a conclusion. In rendering his bill to the patient, the physician takes into account the financial circumstances and charges for his services at a fraction of the tariff rate. Subsequently, the patient appeals for assistance to one of the Benevolent Funds and the doctor receives a communication from the Fund offering a settlement at a reduced rate. What was regarded by the doctor as a normal professional adjustment in dealing with the individual patient takes on a different aspect when a reputedly wealthy Fund enters the picture.

We have the assurance of the Fund directors that they are aware that "in many instances the distressful circumstances of the patient will have led the doctor to submit a reduced bill" and that they are prepared to relate the services to the minimum provincial tariff and offer in settlement usually 50% of this amount. In the absence of a provincial schedule the D.V.A. tariff has been utilized.

The determination of the details of the services actually rendered gives rise to reports and correspondence which have little appeal to the busy doctor and add to his irritation. Practical methods for the reduction of paper work would be welcomed by all.

Medical accounts rarely stand alone as the single indebtedness of a veteran beneficiary and

Canad. M. A. J.
June 1953, vol. 68

an offer of settlement at approximately the same percentage is made to all creditors at the same time. Unless all the creditors agree to accept the settlement offered, the Funds are unable to achieve solution to the entire debt problem and the persistent refusal of one creditor may result in the Funds being obliged to disclaim total responsibility in an otherwise deserving case.

It is my understanding that a recent increase in interest is likely to permit the Army Fund to make offers of settlement to doctors at a rate of approximately 60% of the minimum provincial tariff and this also will be the general practice of the Naval and Air Force Funds.

There is little doubt that the persons assisted by the Funds can be classified as medically indigent and it is only on that account that the profession is asked to accept payment at a reduced rate from these charitable foundations. The individual physician is, of course, entitled to conduct his personal dealings with his patients and these Funds as he sees fit, but it is hoped that this report will assist him to understand their purposes and their limitations. It is encouraging to learn that some doctors formerly outspoken critics of these Funds, now advise deserving and eligible patients to apply for assistance since they have come to recognize them as effective agencies for the relief of economic distress.

HOSPITAL REPORTS

COMBINED STAFF ROUNDS OF THE ROYAL VICTORIA HOSPITAL NO. 8
REGIONAL ENTERITIS

R. D. McKENNA, M.D.,
DOUGLAS WAUGH, M.D.,
R. G. FRASER, M.D. and
H. S. MORTON, M.D., *Montreal*

MEDICAL CONSIDERATIONS

Dr. R. D. McKenna

THE TERM REGIONAL ILEITIS was coined in 1932 by Krohn, Ginsberg and Oppenheim to designate a clinical and pathologic entity. The original description of the clinical features of their cases emphasized the presence of diarrhœa, lower abdominal pain, fever, weight loss and anæmia. Symptoms due to partial obstruction were mentioned, and one of the outstanding features in a number of their cases was the presence of external and internal fistulas from the ileum. The principal physical findings mentioned by the authors in their series of 13 cases were, (a) a mass in the right iliac region; (b) evidence of fistula formation; (c) emaciation and anæmia, a scar of a previous appendectomy and signs of intestinal obstruction. The roentgen study usually demonstrated some degree of obstruction of the terminal ileum, but there was no indication in their paper of any involvement of the cæcum or colon, except for a possible fistulous communication between the ileum and the cæcum. It is really surprising how well this original description of the clinical features as well as the pathological characteristics of regional ileitis has stood the test of time.

However, soon after the appearance of this original article it was learned by Krohn and his confreres that the same pathologic and clinical features of chronic terminal ileitis could be found with lesions anywhere in the mesenteric small bowel. Case reports soon appeared describing identical lesions higher up in the jejunum and ileum, some with segmental areas of involvement, with areas of normal bowel interposed. Quite often too it soon appeared that typical terminal regional ileitis was associated with segmental areas of involvement of the proximal colon. Many terms then became used to describe this disease. Among them were chronic regional ileitis, terminal ileitis, chronic segmental ileitis and ileitis with a segmental colitis, and also ileocolitis.

The disease is rather uncommon. The Lahey Clinic however, have reported on 48 cases studied over a period of 6 years, and the Mayo Clinic on 30 cases seen there in a period of one and a half years. It was actually surprising in reviewing our cases at the Royal Victoria Hospital, to find that we have seen 68 cases in 11 years. The diagnosis in our group of cases was established beyond reasonable doubt by x-ray, surgery or both.

There is apparently no sex predominance. Many writers have commented on the increased incidence among Jewish people. Marshall's series of 29 patients however, included only 3 Jewish patients while 25% of our group were of Jewish extraction. The disease has been reported in most countries. The majority of cases of ileitis seem to come from people in the lower economic bracket, or among those who have recently emerged with considerable struggle from this group. The occurrence among siblings has been reported by Krohn and others and in my short experience I have seen two sisters with this disease. It may occur at any age but the large majority of patients are seen between 20 and 40 years of age.

The pathology and etiology will be discussed by Dr. Waugh. I would like to interpose the suggestion that there are emotional factors of probable important etiologic significance in a considerable number of cases. Unfortunately

however, there have been no large series of cases with adequate psychiatric evaluation and this problem offers a possible fertile field of future study.

The clinical picture is a varied one but there are some five main clinical patterns which may serve as a guide to recognition. (1) Ulcerative Colitis; (2) Intestinal Obstruction; (3) Acute Appendicitis; (4) Nondescript Abdominal Pain, Fever, Weight Loss; (5) Fistula or Abscess. We have seen yet another minor group. There are the rare cases of pyrexia of unknown origin without appreciable gastro-intestinal symptoms which after careful study from every other viewpoint have finally been shown on x-ray study to have regional ileitis. Mixed symptom patterns of course are not uncommon in this disease.

Ulcerative colitis pattern.—Mimicry of ulcerative colitis by regional ileitis and ileocolitis is common and accounted for 21 or 25% of our cases. The major symptom in this group is diarrhœa, and at the onset it may be intermittent, but later, after weeks, months or years may be a continuous affair. Ordinarily this diarrhœa is not severe, perhaps three to four stools a day which are more apt to be mushy or semi-liquid rather than liquid. Tenesmus is not usually associated. Pus and blood in the stool is infrequent and even occult blood may be absent from the stools for long periods of time. Crampy pain is a common complaint, located often in the mid-abdomen or right lower quadrant. Relief of distress by a bowel movement is not as consistent as it is with ulcerative colitis and the pain is often aggravated by the taking of food. This group of patients slowly but progressively lose weight and strength, develop a moderate hypochromic anæmia and evidences of nutritional deficiency. Ileocolitis is more apt to produce this picture than is regional enteritis without colonic involvement. The recognition of colonic involvement in regional ileitis is important from the therapeutic surgical viewpoint.

Intestinal obstruction.—This second group accounts for 10% of our cases. Obstructive symptoms often appear after the ulcerative colitis syndrome has been present for months or years. However, the obstructive picture may appear in the absence of significant antecedent complaints. The diagnosis of obstruction is usually obvious because of pain, vomiting and distension, confirmed by x-ray. It may be impossible in this group to determine preoperatively the cause of obstruction.

Appendicitis.—The third group is that with the pattern of acute appendicitis. In our series, 33 or 43% presented themselves with this picture. In the earlier stages of terminal regional ileitis the signs and symptoms of acute appendicitis may be present, and the differential diagnosis may be made only by direct inspection of the lesion. Some patients also with more advanced chronic ileitis may present with symptoms suggestive of chronic or sub-acute appendicitis. However, in this group careful enquiry will often reveal some history of bowel disturbance or chronic abdominal pain preceding for some appreciable length of time. This group may be distinguished if they are carefully studied.

Abdominal pain.—Pain of some type is present in nearly all cases that have been operated on for regional ileitis. It is often crampy with partial obstruction, or unusually severe with mesenteric or serosal involvement. Pain may be the only complaint and it may be quite nondescript without any definite rhythm, relation to meals or bowel movement. One of our cases, long considered to be a neurotic without organic disease, complained of vague pain and constipation. X-ray studies revealed a very extensive ileal involvement. Unfortunately she has not been greatly benefited by resection and ileotransverse colostomy. I have made some reference to nutritional depreciation but there is an occasional patient in whom the nutritional deficiency pattern will constitute the outstanding clinical feature even though only a small amount of ileum is involved, and there have been a few of these in whom a spectacular cure has been brought about by surgery. There is nothing about the associated fever in this group of cases.

Fistula or abscess.—This group obviously implies some degree of perforation. Free perforation with generalized peritonitis is a rare event, but perforation may occur resulting in localized abscess in either the mesentery or peritoneal cavity with the subsequent development of either internal or external fistulas. An inflammatory mass in the right lower quadrant suggests of course, a number of etiological possibilities, such as appendiceal abscess, right adnexal disease, perinephric abscess, cold abscess, etc., but this also may be due to regional ileitis with walled-off perforation. Internal fistulæ may occur between loops of ileum and ileum, ileum and colon, or they may extend into the bladder, vagina or uterus. Less commonly, perirenal, perirectal abscesses, recto-vaginal fistulas occur. These may rarely present as apparently independent affairs preceding by some years the characteristic symptoms of ileitis.

Other diagnostic aids.—An appendectomy scar seems to have been worn by practically all of our patients. The finding of a movable mass in the right iliac fossa or elsewhere in the abdomen is frequent. Half of our patients show a very pronounced hypochromic anæmia. Weight loss and other evidence of malnutrition are common. Evidences of sepsis, elevation of leucocyte count and sedimentation rate are to be expected. Fæcal analysis shows occult blood in perhaps 50% of cases. Steatorrhœa is a finding in a smaller number of cases particularly where there is extensive ileal involvement. Blood chemistry apart from showing some degree of hypoproteinæmia contributes little to the work-up of these cases.

Diagnosis.—The clinician is completely or almost completely dependent upon the radiologist

Canad. M. A. J.
June 1953, vol. 68

for diagnosis of regional ileitis and ileo-colitis and, this aspect will be discussed by Dr. Fraser. Suffice it for me to say however, that no patient with proved ileitis should be operated on unless a barium enema has been done to exclude involvement of the colon, lest the catastrophe of ileotransverse colostomy be done on an individual with colonic involvement. The differential diagnosis if the involvement is that of a terminal ileum and proximal colon is usually not very difficult. It may be however, confused with ordinary ulcerative colitis, actinomycosis, intestinal tuberculosis, Meckel's diverticulum, and rarely hyperplastic amœbic granulomas. In the absence of pulmonary tuberculosis or the recovery of acid fast organisms from the stool or of sulphur granules from the fistulous tracts, actinomycosis and intestinal tuberculosis are ruled out without too much difficulty. Tumours may be occasionally indistinguishable both from a clinical and radiological viewpoint. We have already discussed the problem of appendicitis, and ordinary ulcerative colitis may be distinguished by the absence of abnormal findings on sigmoidoscopic, and of course positive x-ray findings. Clinical and x-ray differentiation from idiopathic steatorrhœa may be very difficult but at least one can say that stenosis is not present in idiopathic steatorrhœa.

Treatment.—The very nature of the pathological process suggests the frequent necessity of surgical therapy, but the frequent recurrence rate after resection and short circuit operation has resulted in persistence of medical management in some cases. As long as nutritional deficiency, gross anæmia, fistula formation or obstruction have not occurred, it would seem that medical efforts are justifiable. One should certainly make a serious effort to maintain nutrition by high caloric, high protein, low fat, low roughage diet reinforced with multivitamin therapy. Psychotherapy is of definite importance. Although many reports have been discouraging and some of our results poor, at least one of my patients has been maintained in a clinical remission over a considerable length of time with cortisone following upon initial intravenous ACTH therapy. Other good reports with this form of therapy have been reported by Seymour Gray at the Peter Bent Brigham Hospital.

PATHOLOGICAL CONSIDERATIONS

Dr. Douglas Waugh

Regional enteritis may be defined as a relatively uncommon non-specific inflammatory disease characterized by sharply demarcated segmental induration of the wall and mesentery of a portion or portions of the bowel. Where the lesions are multiple the intervening areas of bowel may be quite normal or show only mild inflammatory changes. Whatever portion of the bowel is involved the pathologic features are quite similar in each of the various phases of disease. The principal changes are those of ulceration, œdema and fibrosis, though not necessarily in this sequence nor all together in the same case. In the acute phase the involved area of the bowel is indurated in a rather boggy fashion. It is œdematous and purplish in colour. There may be small amounts of serosal exudate and a small quantity of clear or cloudy fluid in the abdominal cavity. Obstruction in early cases is rare, except in children where the lumen of the bowel is normally small and readily embarrassed by the œdema. There is an acute inflammatory reaction in the mucosa and submucosa. The serosa and muscle are only mildly involved in this or later stages. In these early lesions acute inflammatory changes are seen in lymphatic tissue of the mucosa and mesentary. The latter may be indurated with œdema.

In the subacute phase œdema is even more intense and the bowel wall so turgid that it may fracture when handled. Mucosal ulcers, when present, may be of considerable size and tend to congregate along the mesenteric aspect, though not confined to this area. Large ulcers may coalesce about islands of persisting œdematous mucosa. At this stage the mesentery is more markedly indurated, producing a loss of flexibility which probably accounts for the rarity of intussusception. The inflammatory changes in the lymphoid tissue at this stage tend to be proliferative rather than catarrhal. Early granulomatous lymphadenitis may be distinguished microscopically though this change is more prominent in chronic cases.

The condition of the bowel wall in the chronic phase is that of increasing fibrosis with persistent œdema. Extensive fibrosis of the submucosa causes a great increase in its thickness and gives the bowel a hardness like that of a piece of garden hose. The changes remain sharply demarcated as in the earlier phases. The lymphatic channels in the submucosa and mesentery are widely dilated. Mesenteric thickening is increased by fibrosis and œdema together with an increase in fat content so that the mesentery seems to partially envelop the bowel at its point of attachment.

Mucosal ulcers may be present even in the late stages. The intervening mucosa is usually normal but may now be atrophic. Occasionally the mucosa at the margins of the ulcers shows a polypoid hyperplasia. In the chronic phase the proximal bowel is generally dilated and shows great muscular hypertrophy. At this stage one occasionally sees diverticula in the bowel proximal to the affected segment. It has only recently been noted that mesenteric lymph nodes and lymphoid tissue of the mucosa may contain minute granulomas, similar to those of Boeck's sarcoid or of tuberculosis. These lesions consist of rather poorly localized foci of epithelioid cell proliferation in association with multinucleate giant cells of the Langhans type. These granulomas differ from the lesions of tuberculosis in

that necrosis is absent and the foci are less sharply defined than are the lesions of sarcoidosis.

There is little that is firmly established concerning the etiology and pathogenesis of this disease. Infections of various kinds have been considered. Among the various organisms recovered from the lesions are *Escherichia coli,* anaerobic streptococci and *Aerobacter aerogenes.* However, many cases have yielded no pathogens. Viruses have been suspected but none has so far been isolated. It has been suggested that the disease may be a sequel of bacillary dysentery at a stage in which organisms can no longer be demonstrated. The concept of the disease as a sequel of acute appendicitis is probably unacceptable since many of the appendices removed from these patients are normal. It seems ridiculous to suggest that regional enteritis is a complication of appendectomy. Trauma due to the passage of food particles ought to produce lesions in the upper rather than in the lower small bowel and seems therefore an unlikely etiologic factor.

Psychic disturbances have been considered to play a rôle in the pathogenesis of regional enteritis as has sympathetic hyperexcitability. The presence of large numbers of eosinophils in the lesions of chronic cases has led some to speculate on a possible allergic basis for the disease. Local vascular disorders have also been suggested, whereby a poor collateral circulation within the bowel wall might allow minute mucosal infarctions which then slough and permit a low-grade inflammatory process to become established and perpetuate itself. Such a theory might account for the segmental distribution of the lesions.

Of more interest are the various theories indicting the lymphatic system. One of these postulates rupture of lymphatic channels and the escape of fat-containing chyle. This is then thought to set up a granulomatous reaction in the surrounding connective tissue. Such a process if of sufficient severity and widely enough distributed could conceivably produce the lymphatic blockage and œdema usually seen. A similar theory suggests obstructive lymphadenitis as the initial basis for the disease. Examination of the few early cases available has shown an acute lymphadenitis in which however evidence of obstruction is unconvincing.

On the basis of pathological examinations of 120 specimens Warren and Summers have recently suggested that the basic lesion may be a blockage of lymphatic channels within the bowel wall or in the mesentery. The illustrations in their publication show a proliferation of lymphatic endothelium such as might produce partial or complete obstruction of the affected channel.

None of the foregoing hypotheses concerning the etiology and pathogenesis of regional enteritis has received wide acceptance. The opinion of Pugh that the absence of a known etiologic agent is a prerequisite for the diagnosis of the disease seems to remain the most honest standpoint from which to view this baffling ailment.

RADIOLOGICAL CONSIDERATIONS

Dr. R. G. Fraser

As has been pointed out in the discussion above, if the terminal ileum has been found to be involved in a stenosing, ulcerative inflammatory process, the possible involvement of other segments of small bowel, or of any portion of the large bowel, must always be borne in mind and cannot be too strongly stressed.

In every patient in whom regional enteritis is suspected, therefore, the radiologic examination should include the whole intestinal tract from the duodenum to the rectum. A barium enema will allow not only a complete study of the colon, but usually will permit visualization of the terminal several inches of ileum by spill-over of the barium through a patent ileo-cæcal valve. This can be followed the next day by a barium meal, with periodic examination of each segment of small bowel as the barium progresses through it. Two days will thus usually suffice to examine the whole tract.

The changes observed in the bowel both fluoroscopically and radiographically will depend on the stage of the pathologic process. Although the pathologic anatomy has been discussed in detail brief mention of the changes produced may help to clarify the radiologic appearance.

The pathologic stages generally described are the acute, sub-acute and chronic. In the *acute* stage, the inflammatory reaction in the bowel wall is associated with marked œdema and congestion, with resultant extreme narrowing of the lumen. This may reduce the calibre to no more than two or three millimetres, the area of involvement commonly being demarcated fairly sharply from the normal bowel proximally and distally. Fluoroscopically, spasm and irritability will be notable, and the involved segment will be acutely tender and sometimes palpable as a mass. A very important aspect of this stage will be the preservation of peristalsis, a feature so prominently lacking in later stages. The lumen may even expand to its normal width when a bolus of barium passes through, only to rapidly return to its previously narrowed state. Thus, although the lumen is markedly narrowed by the thickened bowel wall, peristalsis will force barium through, albeit somewhat more slowly than usual, so that obstructive manifestations will not be present, and there will be little or no proximal small bowel dilatation.

It is in this stage that a diagnosis of acute appendicitis is commonly made and the patient operated upon for that, without referral for radiologic examination.

Although minimal mucosal ulceration may occur in the acute stage, it is not until ulceration

Canad. M. A. J.
June 1953, vol. 68

becomes fairly extensive that it is radiologically demonstrable, therein lying the chief difference between the acute and *subacute* stages. The fairly regular narrowing seen in the acute stage is thus replaced by marked irregularity of the barium-filled lumen, with distortion of the mucosal pattern and obliteration of the normal markings of the plicæ circularis (Fig. 1). Spasm and irritability will be less prominent features than in the acute stages, but residual pliability of the bowel wall will still allow peristaltic waves to pass through the involved segment. Ulceration may be so extensive as to lead to perforation and the development of sinus tracts or fistulæ, either to the skin surface or to adjacent loops of small or large bowel. Frequently these will be demonstrated by barium passing along their length. The majority of cases referred to the Department of Radiology, Royal Victoria Hospital, fall into the subacute group, and undoubtedly this is true of other centres as well.

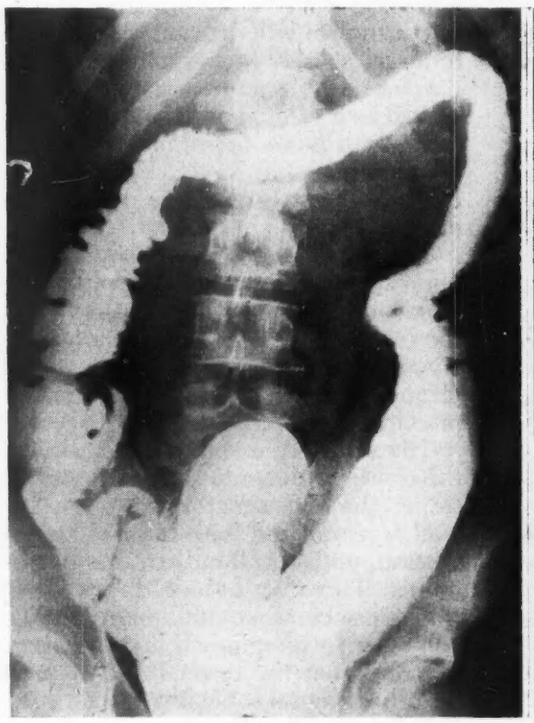

Fig. 1

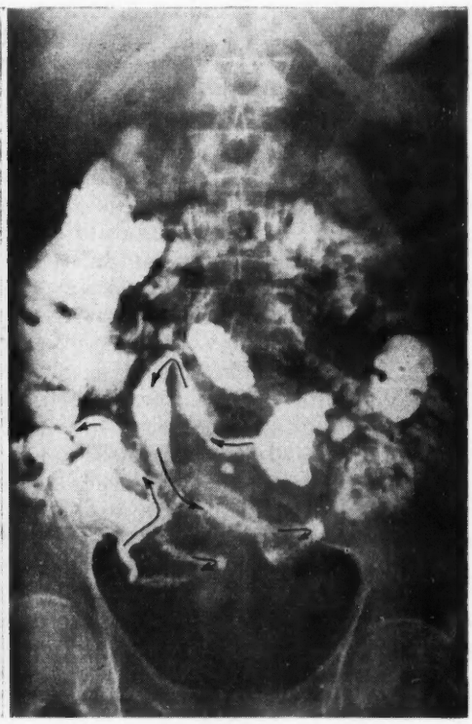

Fig. 2

The *chronic* stage is characterized by progressive fibrosis. Whereas in the previous stages, a certain amount of pliability of the involved segment has been present, there is now no semblance of motility or plasticity, and the end result is a rigid, irregularly and persistently narrowed tube (Fig. 2). Frequently the involved segment can be palpated as a sausage-shaped tender mass. Peristalsis is completely absent, and it is this loss which leads to the development of subacute, and sometimes acute, small-bowel obstruction. Small-bowel dilatation, with ileal stasis, will then be prominent features of the radiologic picture.

It is to be noted that a generalized disturbance of small-bowel motility is an almost constant feature in all stages of regional enteritis. In the acute and subacute stages, this usually takes the form of hypomotility, clumping of barium, and localized spasm and dilatation (Fig. 2). In the chronic stage, the typical changes of mechanical ileus, either subacute or acute, will be observed in almost every case.

Since the terminal ileum is the most commonly affected segment of small bowel in regional enteritis, involvement of the cæcum is to be expected in a fair percentage of cases. This may take the form of spasm and irritability, secondary to the ileal disease, or to actual organic change in the cæcal wall, or to a combination of the two. If spasm is the predominant feature, extreme difficulty will be encountered in filling the cæcum with barium, its irritability maintaining it in a constant state of contraction. Barium will seem to "skip" from the terminal ileum to the ascending colon, merely coating the mucosal surface of the cæcum with a thin layer.

If organic involvement of the cæcal wall occurs, precisely the same changes are present as are seen in the small bowel and other portions of the colon. There is irregular persistent narrowing of the lumen, and marked distortion of the mucosal pattern with ulceration. The line of demarcation from the uninvolved colon distally is usually quite sharp.

The frequency of recurrence following inadequate resection of the diseased bowel is commented upon and stressed in the section on the surgical aspects of the disease. The radiological

features of such recurrence do not differ sufficiently to warrant further description. The region of the anastomosis is, of course, the area most frequently involved.

Two rather unusual features, not commonly described by others, have been observed in four of the twelve cases of regional enteritis studied in our Department since January 1950. In two patients, definite irregularity and distortion of the mucosal pattern of the duodenum was observed, in addition to the abnormality of the terminal ileum. The involvement was apparently localized to the duodenal cap and the proximal half of the 2nd portion in each. Irritability, spasm and tenderness were observed fluoroscopically. A striking absence of duodenal involvement in regional enteritis has been remarked upon by Sproul and Golden. It is noteworthy, however, that our evidence is purely radiological, only one of the two cases having been operated upon, with vagotomy and gastroenterostomy being the only procedures undertaken. However, the evidence is believed to be sufficient to warrant a fairly positive diagnosis of localized duodenal enteritis.

In two other cases, incomplete descent or rotation of the cæcum was observed. In so small a series, this frequency cannot be considered as indicating any definite cause-and-effect relationship between the organic lesion on the one hand and the congenital anomaly on the other. The association is interesting to record, however, on the chance that other observers have noted it in the past or may note it in the future.

The radiological differential diagnosis must include, first and foremost, alimentary tract tuberculosis, in view of its common involvement of the cæcal region, including the terminal ileum. If the ileum alone is involved, non-tuberculous inflammation is much more likely, since cæcal involvement almost always accompanies or precedes tuberculosis of the distal ileum. In many cases, differentiation will depend in final analysis on the presence or absence of pulmonary phthisis. Ileo-cæcal actinomycosis may be impossible to differentiate without the demonstration of "sulphur granules" in the fistulous tract. Primary carcinoma, or lymphoblastoma, involving the terminal ileum may produce changes indistinguishable from regional enteritis. Such conditions as intestinal allergy or non-sclerosing ileitis (Golden) may induce changes strikingly similar to regional enteritis, but confusion will not be too likely to occur if the clinical history is given sufficient weight in the final evaluation.

SURGICAL CONSIDERATIONS

Dr. H. S. Morton

This is a review of cases of regional enteritis which have been treated in the Royal Victoria Hospital during the past ten years. Fifty-three cases are included in this series with no operative mortality.

A classification of the various terms used in enteritis will help materially in understanding this rather complicated clinical picture.

(a) Acute regional ileitis. (b) Entero-colitis; involving both the small and large bowel. (c) Regional colitis. (d) Acute jejunitis which is an acute severe disease frequently fulminating.

This may involve "skip areas" from the ileum to the duodenum inclusive.

Regional enteritis is a completely non-specific disease, and the term is used as a "waste paper basket" to include almost any inflammatory process except tuberculosis. The types are:

(1) Acute regional ileitis which is the commonest form of the disease. It has as a complication the appendiceal form in which the cases present as acute appendicitis and normally have an appendectomy performed. (2) The diarrhœa or straight enteric group. (3) Those presenting an intestinal obstruction. (4) Lastly, the fistulous group. The fistulæ may be either internal going to other loops of the bowel or even to the bladder, or external leading to the skin.

A third division of these cases may be made on clinical grounds into acute, chronic or fulminating.

Of the 53 cases, 23 (43%) had a pre-operative diagnosis of acute appendicitis, and the average length of illness before operation was two days. In all save two the appendix was removed. Clinically this appeared to be normal, but the pathologist usually reported exudative, proliferative appendicitis. There was only one case that had purulent appendicitis.

There has been considerable debate on the advisability of removing the appendix when at operation regional enteritis instead of appendicitis is encountered. The follow-up on these cases shows that the majority of the appendicular forms have no further trouble, while half of the cases that have short circuit or resection did not have a previous history of appendectomy. It would, therefore, be unwise to blame the removal of the appendix for subsequent complications. To miss one case of purulent appendicitis would be unfortunate. Therefore, the recommended treatment for this group is removal of the appendix. It is important to record that the mesenteric glands were enlarged in 90% of all the cases.

The obstructive group, though small (7%) is very important from the surgical point of view as it demands prompt attention. An interesting case has been reported in which narrowing by fibrosis was so marked that a single pea caused complete obstruction.

Fistulæ are slightly more common and account for about 10% of the cases. The most important differential diagnosis is actinomycosis of the right iliac fossa. When the clinical pictures are compared there is absolutely no difference, and only the finding of the characteristic ray fungus of actinomycosis establishes the diagnosis.

Treatment.—In the more acute forms of regional ileitis the recommendations for treatment

Canad. M. A. J.
June 1953, vol. 68

have passed through three stages since Krohn published his paper in 1932. The first phase was the cautious advice to short-circuit the lesion. Then resections were advocated, with a resultant increase in mortality. Now the Mount Sinai school practice the exclusion short circuit operation. There are points in this procedure to be emphasized. The site of the anastomosis is made at least two feet proximal to the apparent disease, because it is notoriously difficult to determine the level of mucosal involvement from the serosal aspect. By exclusion is meant dividing the small bowel distal to the anastomosis, leaving a blind pouch, and thus putting the diseased area at rest.

Garlock *et al.* reported the long term results of this treatment in 57 cases, with 6 recurrences, and compared the exclusion operation with resection. In the chronic form of this disease it is better to excise the affected bowel. This involves a right hemicolectomy and ileo-transverse anastomosis, the site being 2 feet proximal to the apparent disease.

In our series, 9 cases had short-circuiting operations and 12 had resections, of which 8 were primary resections, and 4 were done after short circuiting of the primary resections. One had 3 resections and two had 2. Eight cases had laparotomy only.

The severe fulminating type is more common when the jejunum is involved. This is best described as having "skip areas" with apparently normal bowel between diseased ileum, jejunum and even duodenum. There was one such case in this series, and the surgeon, finding it difficult to determine the limits of mucosal involvement and exactly what areas of the small bowel to remove, performed a sub-diaphragmatic vagotomy. This case has been followed for two years and is well. This contrasts with the treatment of acute jejunitis reported by Brynjulsen of resection, with a mortality of 87%.

In the more chronic forms of jejunitis associated with ileitis, the question of how much of the small bowel can be removed satisfactorily frequently arises. Although many people state that not more than 6 feet of the small bowel should be removed, there are two recent cases in Montreal in which all but 2 feet in one and 18 inches in the other have been successfully resected. There are some nutritional difficulties, but these cases demonstrate that extensive removals are possible. However, they should only be performed in chronic cases.

Regional enteritis, therefore, is a disease with many manifestations. In the commonest and mildest form it is best treated by a simple appendectomy. The severer and more acute type usually responds to the short-circuit exclusion operation, while the more chronic severe forms require resection. When the jejunum is involved, especially in severe cases, vagotomy is the treatment of choice.

CLINICAL AND LABORATORY NOTES

TABLET CONTAINER CHILDREN CANNOT OPEN

D. F. CROSS, M.D.,* *Montreal*

"Villainous poison lies concealed in sweet honey"—OVID.

WITH ITS ELEGANT and palatable pills, tablets and "medicated candies" the drug industry has largely succeeded in rendering "bitter pills" an anachronism. But these modern medical presentations, sugar-coated, often flavoured and brightly coloured, have brought a new and growing danger into the home. As Ryan[1] puts it;

"Recently there has been an increasing tendency on the part of pharmaceutical companies to produce medicaments which pander to the puerile palate so that, where formerly children needed a great deal of patient cajoling in the administration of medicines, now we find them imbibing freely and sometimes demanding an array of pleasantly and brightly coloured sugar-coated tablets. Thus it is easy to realize how potentially dangerous, as regards overdosage, some of these substances can be."

It is obvious that the danger is greatest to children of pre-school age. Their natural instinct is to put everything to gustatory test, and the exploration of the medicine cabinet, or of a mother's handbag, becomes more and more tempting as the pills and tablets they may contain are presented in ever more attractive forms.

In connection with the high incidence of accidental poisoning of children by ferrous sulphate tablets, Thomson[2] in 1947 and again in 1950, sounded a warning. "It would seem to be urgent that *administrative action* should be taken now to prevent these useful tablets being dispensed in unsafe containers . . ." and made an urgent plea to secure really safe packing and storage of these pills when dispensed.

Sooner or later every general practitioner is faced with an emergency summons to a child victim of accidental drug poisoning. During 1951 in U.S. hospitals, alone, nearly 320,000 children were treated on this account and, according to reports in the medical press, the incidence of such accidents is steadily rising all over the world. It is estimated that 1% of such cases prove fatal.

Craig[3] in 1952 reported and surveyed 482 cases of accidental poisoning in childhood in two Scottish hospitals. Again, of all children in the emergency ward of an Oakland (California) hospital during the period of 1945/49 nearly 27% arose from the child gaining access to medicinal tablets, etc., in the household.[4]

Analysis of the age groups of 244 children under 13 years of age suffering from accidental poisoning and admitted to a Children's hospital in Brisbane (Australia) is most revealing.[1]

*Senior Resident, Alexandra Children's Hospital, Montreal.

Space prevents the inclusion of other statistics, but what emerges clearly from the above as well as from the study of further data, is that the danger years are under 5; *the peak incidence being among toddlers between 2 and 3.*

Another interesting and important point comes to light when we consider the nature of the drugs concerned in the great majority of these cases; for the most part they are within the category of relatively innocuous household remedies. Such things as ferrous sulphate, Blaud's pills, laxatives and analgesic combinations of A.S.A. which are harmless in normal doses both to adults and children, proved the commonest lethal agents to young children consuming them in quantity.

It is idle to blame parents and nursemaids for these tragedies, and urge greater care; these are counsels of perfection. *Quis custodiet ipsos custodes!* Everyone is at times forgetful, and more of us are becoming increasingly so by reason of the breath-taking pace of modern life. A locked medical cupboard well out of the toddler's reach is effective only if the drugs are

Years of Age	*Percentage of Group*
1st	3
2nd	47
3rd	29
4th	9
5th	5
6th to 13th	7
	100

invariably kept in it; but it is interesting to note that most accidents are due not so much to the child *opening* the container but because cap or lid become mislaid or lost, so making free the contents to the inquisitive little explorer.

The numerous suggestions for promoting safety include the mixing of a bitter-tasting substance in the tablet coating, the addition of an emetic, and the packaging of medicaments in units containing as many individual doses as would be unlikely to harm a child. Bitterness, however, has little or no deterrent action on toddlers, whose taste-buds are at this stage seldom well developed. The inclusion of emetics in all tablets, to make either adults or children vomit when taking excess dosages is, of course, wholly impracticable; response to emetic drugs varies among individuals within very wide limits, to state one reason only. Also, as a matter of simple economics, the packaging suggestion cannot be seriously entertained. The crux of the problem has been summed up in a terse and admirable phrase;[5] *"Some obstacle should be placed between the tablet and the child".*

What, therefore, has been long needed is a safety container as fool-proof and "kiddie-proof" as human ingenuity can devise and which, in fact, *does* place "an obstacle between the tablet and the child". The writer's attention has recently been drawn to a container which appears to answer this need and which is here illustrated (Fig. 1). The principle of its design is such that a small child is unable to open it; moreover the lid, being undetachable, cannot become mislaid.

This container constitutes an entirely new approach to the problem, and it carries the hallmark of that simple ingenuity which is the essence of genius. It consists of an aluminum tube fitted with a snap closure which remains attached to the body of the container even when it is opened. By following the simple instructions printed on the lid, any adult can readily gain access to the medicaments enclosed. *But no normal child of under five,* or in many cases up to 8-9 years of age, even if sufficiently ingenious to solve the puzzle for himself, would have either the hand-span or sufficient strength of finger to open the lid.

In our hospital we have made repeated trials on individual children, and numerous observations on groups, varying greatly in physical strength and I.Q. Some of the containers were

Fig. 1

empty and others fitted with candies in the presence of the kiddies to excite their greater interest. In the above-mentioned age range, not one in a hundred children succeeded in opening the container, even if previously shown how to do it. I understand that results obtained in England are strikingly confirmatory of these findings.

In brief, I believe the "Kidipruf" container to be new in principle, hygienic, amazingly simple in handling and almost 100% safe at pre-school age. It is hoped that pharmaceutical houses will soon package all their tempting medicinal pills and tablets in these containers, and that they will be available also to mothers and guardians in the not very distant future, from retail druggists.

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Canad. M. A. J.
June 1953, vol. 68

The Canadian Medical Association Journal

published monthly by

THE CANADIAN MEDICAL ASSOCIATION

Editor: H. E. MACDERMOT, M.D., F.R.C.P.[C.]

Editorial Offices: 3640 UNIVERSITY ST., MONTREAL

(Information regarding contributions and advertising will be found on the second page following the reading material.)

EDITORIAL

A CANADIAN COLLEGE OF GENERAL PRACTICE

It is not easy to summarize briefly discussions of a committee which extended over the best part of two days and were taken part in by about twenty men. But certain impressions may be given regarding the recent meeting of the committee to explore the matter accrediting general practitioners.

As was right, the history of this movement in Canada was given considerable prominence. It extends over the last five years, but it was pointed out that the Royal College of Physicians and Surgeons of Canada had a gestation period of at least nine years. On the other hand, the American Academy of General Practice, and the more recently formed College of General Practitioners in the United Kingdom have both been developed in a comparatively short period. So that on the one hand we in Canada may be reassured in not being precipitate, but also have before us examples of more speedy accomplishment.

The practical bearing of these comparisons is that there are tendencies amongst some of our profession to seek the solution of our delay in affiliation with the American body. Certainly this is not the feeling amongst the majority, but it is definite enough to call for consideration.

Two main questions stood out. (1) Why should we have an organization of general practitioners such as is proposed? (2) Through what avenues should such a body be set up?

The chairman, Dr. Murray Stalker, said at the outset that the ultimate object was to improve the status of the general practitioner, and at the same time to maintain the unity of the profession; and that was an excellent epitome of the situation.

In improving the status of general practitioners and so maintaining a high standard of medical care, it was generally agreed that education should be given first place. The many channels through which this would be carried out were fully discussed; postgraduate teaching; research; hospital appointments, etc. And then, following naturally on this was the need for a body to carry on the work.

The choice of a suitable title for such a body took some time, but finally "The College of General Practitioners of Canada" was agreed on. It was felt that this should be an organization which should stand on its own feet, but should be closely allied to its parent, the Canadian Medical Association. It was hoped that the leadership of the C.M.A. in developing the Royal College of Physicians and Surgeons might serve as a precedent in developing the new College. Membership in this would be dependent on membership in the C.M.A. and therefore would tend to strengthen it.

A recommendation was therefore made to General Council of the Association that the proper machinery might be set up to bring about the formation of such a body. The procedure suggested was that a nucleus committee might be appointed by the C.M.A. to carry out the project in consultation with the already existing Section of General Practice and the Executive Committee of the Association.

The exploratory committee has cleared the way admirably. Their recommendation will now come before Council, and if solicitude for high standards has its reward we may look for a start to be made on the establishment of a College of General Practitioners in Canada.

THE NEXT STEP TOWARDS A NATIONAL HEALTH SERVICE

There may seem to be a lull in the winds of discussion over health insurance. But it must be plain that this can be no more than a pause in developments. If there is less public discussion about dealing with the cost of illness it certainly is not because this has become less of a problem. The spread of prepaid medical care plans may gradually be having some effect in showing people that they can insure themselves against the cost of illness. But of course this so far applies only to the minority. To most people the crippling economic possibilities of illness are still terrifying.

The expressed view of the Association is that we recognize the necessity for some plan by which the cost of illness can be so spread as to remove its burden. We have also committed ourselves to exploring the possibility of so co-ordinating existing prepaid medical care plans that these shall take up the protection of a large part of the population. The obvious defect in these prepaid plans is that they cannot help the indigent, or even those in the lower income brackets. Relief for these can only come from the government.

So then our plan so far would be partly dependent on individual initiative and partly on government aid. Whether this would be a possible arrangement, even if it were desirable, has not yet been examined. Certainly no definite proposal has been put forward.

Would the government listen to any plan proposed by the medical profession? Have we any plan to propose, apart from the joint arrangement already mentioned?

There are those that believe that Ottawa would welcome some considered plan by our Association. That at once would place the responsibility of leadership on us. This brings us to a difficulty which so far has been insuperable. In spite of the prolonged discussions that have taken place over the last twenty years and more, our Association through its general council has never been able to agree as to the best course to pursue. So that in effect no other attempt has been made to alter the present state of affairs, other than the formation of Trans-Canada Medical Service.

We have no assurance that the government will take up the cost of medical care for those unable to afford prepaid medical protection, rather than put into force an overall insurance scheme. There was a time when such a scheme seemed to be imminent. Circumstances slowed down these plans, and then we witnessed the British all-inclusive scheme of free medical care. Now that this form of national health service has weathered the storms of criticism and it has been shown that it is practicable—if expensive; and has desirable aspects—if some that are less desirable; it is reasonable to expect our own government to examine the situation again. We have no reason to believe that an over-all scheme of health insurance will not be brought forward which we shall have to accept in the same way that the profession in Great Britain was forced to accept the present national health services.

NEXT — WINNIPEG

This is the last issue of our Journal before the Annual Meeting. By now we have all read the Program that has been planned for this, our Annual get-together.

There are some grave issues before us at this time that may and can have lasting effects on everyone in our profession. A united effort is always the best assurance to success in settling these issues. The professional and scientific parts of the program give promise of an excellent refresher to everyone, whatever his or her chief interest. Lastly, Winnipeg, famous for its hospitality, will assure wives as well as husbands an enjoyable interlude. We do hope this meeting will set a new record. See you in Winnipeg!

A.H.N.

Editorial Comments

THE PROBLEM OF REGISTRATION OF ALIEN PHYSICIANS IN ONTARIO

It is well known that each Province controls its own Registration of Physicians. Perhaps it is not generally realized what problems are involved in the exercise of this control. In no province are these better exemplified than in Ontario. It is an experience well worth while to witness the Meetings of the Committee on Education and Registration appointed by the College of Physicians and Surgeons of Ontario to pass on the qualifications of graduates of foreign Medical Schools who wish to practise in that province.

Up to ten years ago the work of this Committee was light. The College had to deal only with the routine registration and perhaps a few cases of physicians from outside Canada. After the war large numbers of physicians from Europe were forced to seek refuge and many arrived in Canada. At first some of these were men of well established standing, sometimes even of international reputation. Many were from the best medical schools on the continent and their standing was relatively easy to appraise, nor, it may be added was it so difficult to absorb them during a period of acute shortage of medical personnel in our own country.

Then the picture gradually changed. Men and women with medical training gained only during the war years began to present themselves for registration. The quality of their training had to be much more carefully appraised. They were not "displaced persons" but had been trained in the post-war period.

Out of this grew the screening process now in operation by this Committee. Actually the Committee is not granting licenses. It is only determining whether the applicant shall be granted

the right to take the examinations of the Medical Council of Canada; the right being embodied in the so-called enabling certificates issuable by the Province. The candidate's application is considered (and may be held in abeyance) a long time before he is asked to appear before the Committee. A special letter informs him of what he must do. He must have had a year's rotating internship in an approved hospital; must be a Canadian citizen or formally declare his intention to become one; must have papers in support of his claims to training, etc. The Registrar in most cases has a personal interview and reports are also asked for by way of a questionnaire from those with whom the candidate has worked.

This constitutes a reasonable amount of evidence as to the worthiness of the candidate. Even so the Committee may find it difficult to judge the merits of a given case. Sometimes an inability to speak English fluently makes it necessary to have the candidate take another year's preparation in this respect. The Extension Department of the University of Toronto has arranged classes for the teaching of English, especially of a Medical and Biological nature. A candidate may not show evidence of enough knowledge of the Basic Sciences, and further training in this is advised. Satisfactory examinations have to be passed both in English and the Basic Sciences.

It is impressive to watch this Committee at work. The amount it has to do may be realized from the fact that there were 106 applicants this spring, and as no more than 25 can be dealt with properly in one day it means four days of devoted labour. The Committee meets twice yearly. The problem is to deal fairly with these people and at the same time protect the public, as far as may be possible, by maintaining high standards of medical training. The patience and care with which the work is carried on is deserving of high admiration. It may seem that the province of Ontario alone is concerned with these particular investigations but in point of fact they have implications of a national aspect.

DR. FRANK S. PATCH

It is with the deepest regret that we record, on the eve of going to press the news of the death of Dr. Frank S. Patch, a former President of our Association. Full details will appear in our next issue.

MEN AND BOOKS

THE FIRST PHYSICIANS IN NOVA SCOTIA

KENNETH A. MacKENZIE, M.D., C.M.,
Halifax

THE FIRST PHYSICIANS in Nova Scotia were the "medicine men" of the Indians. They used many vegetable and animal products in an empirical manner. Their methods were highly coloured by magic and superstition. One observation only is worthy of record; the infusion of fresh leaves, especially spruce, was found to be useful in the prevention and treatment of scurvy.

The first European physicians came with the adventurers from France. The apothecary and barber-surgeon were desirable members of many crews. It is on record that John Cabot had in his crew a barber surgeon, and if, as some historians claim, he landed in Cape Breton, this surgeon would be the first to set foot on American soil. It is also on record that Jacques Cartier had with him an apothecary, Francois Guiltault, to "look after the health of his crew".

The first trained physicians in Nova Scotia, then called Acadia, came with de Monts in 1604. After exploring the coast of Acadia, including Port Royal, de Monts crossed the Bay of Fundy and built his settlement at St. Croix. Here his colonists suffered intensely from the severe cold and especially from a new disease later known to be scurvy. Thirty-five of his party of 79 died. Many others were extremely ill. In the spring of 1605 the remainder, 45 in number moved to Port Royal where they found the climate more pleasant.

In de Monts' party there were four physicians, Louis Hebert, Master Stephen (Etienne), Daniel Hay and I. M. P. Deschamps. Some historian has made the claim that Daniel Hay was the first doctor to practice in Canada but this claim does not seem clear. Hebert and Stephen were certainly at St. Croix and all four were, at some time or other, at Port Royal. There is therefore some difficulty in claiming priority for any one of them. We are indebted to Champlain for interesting records of his physicians and surgeons. The following extracts from Champlain's Narratives, copied from an article "Acadian Medicine" by the late Dr. W. H. Hattie and published in the Nova Scotian Bulletin 1926, indicate a remarkable degree of efficiency in the doctors of that period.

CLINICAL DESCRIPTION OF SCURVY

"During the winter, many of our company were attacked by a certain malady called Mal de la Terre; otherwise scurvy, as I have since heard from learned men. There were produced in the mouths of those who had it, great pieces of superfluous and drivelling flesh (causing extensive putrefaction) which got the upper hand to such an extent that scarcely anything liquid

could be taken. Their teeth became loose and could be pulled out with the finger without causing pain. The superfluous flesh was often cut out causing them to eject blood through the mouth. Afterwards, a violent pain seized the arms and legs which remained swollen and very hard and all spotted as if with flea bites; and they could not walk on account of the contractions of the muscles, so that they were almost without strength, and suffered intolerable pains. They experienced pain, also, in the loins, stomach and bowel and had a very bad cough and were short of breath. In a word, they were in such a condition that the majority of them could not rise nor move and could not be raised to their feet without falling down in a swoon. So that out of 79 who composed our party, 35 died and more than 20 were on the point of death. The majority of those who remained well also complained of slight pains and were short of breath. We were unable to find any remedies for those maladies. A post mortem examination was made of several to investigate the cause of their malady."

POSTMORTEM REPORTS

"In the case of many, the interior parts were found mortified such as in the lungs, which were so changed that no natural fluid could be perceived in them. The spleen was serous and swollen. The liver was woody and spotted, without its natural colour. The vena cava, superior and inferior, was filled with thick coagulated black blood. The gall was tainted. Nevertheless, many arteries in the middle, as well as the lower bowels, were found in a very good condition. In the case of some, incisions with a razor were made on the thigh where they had purple spots, whence issued a very black clotted blood. This is what was observed on the bodies of those infected with the malady. Our surgeons could not help suffering themselves, in the same manner as the rest. Those who continued sick were healed by Spring, which commences in this country in May. That led us to believe that change of season restored their health, rather than the remedies prescribed."

BIOGRAPHICAL NOTES

Louis Hebert (1565-1627) was born in Paris, the son of an apothecary to the Queen, Catherine de Medici. Louis, who inherited property from his father was in comfortable circumstances, trained as an apothecary and destined to occupy a prominent place in his native country. However, captivated by stories of the New World, he forfeited the comforts of home to seek his fortune in a new land. At the age of 40 he came with de Monts to St. Croix in 1604 and moved to Port Royal in the following spring. He endured many hardships. Atlantic crossings were slow and dangerous sometimes taking two or three months. The mortality from disease was terrific, 35 deaths at St. Croix and 12 at Port Royal. The Indians were hostile and some colonists were murdered. Food supplies were inadequate and famine was narrowly averted by the use of meat, fish, fowl, wild berries and nuts. In 1607 Hebert reluctantly returned to France and returned to Nova Scotia with his wife in 1610. Madame Hebert was the first white woman to come to the New World and she was joined a little later by Madame Poutrincourt. In 1613 the colony was destroyed by Argall and the French sailed for France. In 1617, Hebert came with Champlain to Quebec where for the remainder of his life until his death in 1627 he devoted his energies to agriculture. He is regarded as the father of agriculture in Canada and his name has been preserved for all time by a monument in the City of Quebec and a commemorative tablet at Annapolis Royal.

Hebert's claim to fame rests mainly on his contribution to agriculture, but he was first of all a physician. Apothecaries in the old days were botanists always on the alert for something new. Hebert explored the hills, valleys, woods and river banks of the territory in the vicinity of Port Royal studying new plants and hoping to discover in them some medicinal virtues. His interest in plant life led him to the study of food products. He was the first to grow wheat and was able to demonstrate the great fertility of the soil for wheat and other foods. His attempt to cultivate wild grapes did not meet with success. Hebert studied the symptoms of the serious disease, later known as scurvy, and used many remedies without success. He administered physical and mental help to colonists and natives according to what knowledge he had. He learned the Indian language and became friendly with the savages. He nursed Membertou, the great Indian Chief, age 100 during his last illness, a tribute to the confidence which the natives had in him.

With Daniel Hay, he was a member of the "Order of Good Cheer", (L'orde de Bon Temps) the first social club in Canada. This club was organized partly as a health measure as it was learned by observation that those who lived an out-door life and spent time in hunting and fishing, did not develop scurvy as readily as those who confined their work to the duties of the Fort.

Master Stephen (Etienne) is mentioned by Champlain as one of his surgeons. He probably conducted some of the post mortems described in Champlain's Narratives. He was present at Port Royal but was not a member of the Order of the Good Cheer. He was a barber surgeon and may have held a lower rank than Hebert and Hay.

Daniel Hay, surgeon apothecary came out with de Monts. He was a member of the Order of Good Cheer. He returned to France and there is nothing definitely known about his subsequent career.

I. M. P. DesChamps was a surgeon from Honfleur and nothing is known about him except that he was at Port Royal.

This is about all that is known of the first four physicians in Nova Scotia. For the next hundred and fifty years there is very little of medical interest to record.

Twenty years after the destruction of Port Royal by Argall M de Razilly brought sixty French families to Acadie and settled them in the vicinity of Port Royal. These, with others who came later were the ancestors of the French Acadians, who, at the time of the expulsion (1755) numbered about ten thousand. They were an in-

dustrious people and took full advantage of the rich alluvial soil adjoining the Bay of Fundy and they added to their acreage by building extensive dikes after the pattern with which they were familiar in the home land. In time they raised a variety of crops and stocked their farms with domestic animals and were able to export farm produce as well as look after their own needs.

A population such as this would be expected to have some medical services but there is very little recorded. A few names have been preserved; Armand Bugeaud and Mouton at Mines, Horton, Sainte Cenne at Annapolis in 1731, Surgeon St Jus with the French troops at the Battle of Grand Pre, 1747. No doubt there were many others unnamed. The best known surgeon of the French period was Jacques Bourgeoise (1621-1701) brother-in-law of the Commandant Doucet de la Verdure. Aulnay refers to him as "his Surgeon" but his place in history is due to his political activities, first at Port Royal and later at Chignecto. In 1654 he was held by the British as a prisoner hostage and his signature was attached to the articles of capitulation. In later years he with his family took a prominent part in the commercial and agricultural development of Chignecto.

At Louisburg, not yet a part of Nova Scotia, the population was estimated from 4,000 to 10,000 and sometimes more. They had many doctors but only four names are available to me at the present time; Vreman, Le Grange, LeRoux, and Bertin. Le Grange, chief surgeon to the garrison, first at Port Dauphine, later at Louisburg is said "to have confined his skill to his razor and lancet". In 1735 he returned to France on account of ill health and died the following year. His successor as chief surgeon was his son-in-law Bertin who held this appointment until the fall of Louisburg 1758. LeRoux was an apothecary and compounded all his own medicines.

Before the founding of Halifax in 1749 the English made no serious attempt to encourage settlers in Nova Scotia. The only medical men were those attached to military or naval units. One name only appears in the records, Dr. William Skene, who probably came with Nickolson's forces in 1710 and was garrison surgeon at Annapolis from 1713 to 1749. He held several non-medical appointments; member of His Majesty's Council from 1720 to 1749, justice of the peace in 1727—date of organization of this office. In 1737 he was one of the commissioners appointed to settle the boundary dispute between Massachusetts and New Hampshire and in 1742 a similar dispute between Massachusetts and Rhode Island. For some unexplained reason he was not present when Cornwallis landed and nothing is recorded about his subsequent career.

In order to understand in some measure the scarcity of records regarding the work of doctors in the old days one must try and picture conditions as they were during those periods. The work of medical men was greatly limited partly by the imperfect state of medical knowledge and partly by the small number of patients. The surgeon could dress wounds, set fractures, use a lancet to open an abscess or do a venesection, still a recognized remedy, or he might have to do an emergency amputation. Medical illnesses while severe and often fatal were not understood and the doctor used empirical remedies on a plane not much above the level of the "medicine men". Obviously the doctors had much spare time, and they were called upon to do various menial duties. One definite duty of the surgeon was barbering. An interesting note which I recently came across said that "the priest had forbidden the physicians to cut hair or shave on Sundays or Holy days and the punishment for this sin was to be denied absolution". Some physicians signed on for multiple duties, for example, physician and fisherman or physician and cook. As the real duties of the physician have been kept out of the records it may be fortunate that the records of menial duties have also been withheld.

A few extracts from records throw some light on the standing of doctors. In the personnel of General March's expedition in 1707 the five chaplains are listed with the officers with full names, home addresses and denomination. At the end of the list appears the following: "doctors and mates; bombardeers and cannoneers; armourers; commissaries". No names are given. A report dated 1750 is interesting: "Chaplain's salary $600.00. Surgeon's salary $250.00" (this was paid in English money).

There are many references to physicians in most of the old historical records. The work which they have done is mentioned but seldom is a name mentioned. One instance of this may be given: Governor Brouillan died at sea on board the King's ship *Le Profond* in 1705. His heart was taken from his body, placed in a casket and buried with suitable honours at Annapolis. His body was buried at sea. The name of the surgeon who performed this important duty is not mentioned.

This period of early medical history came to an end when a number of physicians came with Cornwallis in 1749 and a few years later some well trained men came from the New England states. Some of these came before the Expulsion and are known as pre-loyalists. During the revolutionary war a large number came as refugees and many remained.

It is a great mistake to think that because you have read a masterpiece once or twice or ten times, therefore you have done with it. . . . You ought to live with it and make it part of your daily life.—John Morley.

ASSOCIATION NOTES

SOME HIGHLIGHTS OF THE ANNUAL MEETING

In addition to a full program of business and scientific sessions, which have already been reported, the 86th Annual Meeting at Winnipeg provides some noteworthy events which are summarized below. Members and their wives are requested to make a note of these features, and to plan to attend as many as possible.

Monday, June 15.—Meetings of the General Council and affiliated specialist societies.
5:00 p.m. Reception and tea for members of the General Council and their wives, by His Honour the Lieutenant Governor of Manitoba, R. F. McWilliams.

Tuesday, June 16.—Meetings of the General Council and affiliated specialist societies.
6:00 p.m. Medical Exhibitors Cocktail Party.
7:00 p.m. Dinner to the members of General Council and their wives, tendered by the Manitoba Division.

Wednesday, June 17.—Scientific sessions.
8:30 p.m. Annual General Meeting—installation of the President.
10:00 p.m. President's Reception and dance.

Thursday, June 18. Scientific Sessions.
12:30 p.m. Buffet Luncheon, Armed Forces Medical Section. Everyone is invited.
8:30 p.m. Special performance of the Royal Winnipeg Ballet, Playhouse Theatre. This promises to be an unusually delightful event, and members are urged to reserve their tickets as early as possible.

Friday, June 19.—Scientific Sessions.
12:30 p.m. Associated Luncheon. Speaker, the Hon. Paul Martin, Minister of National Health and Welfare.

An opportunity will be afforded for visitors to Winnipeg to inspect the new building which houses the Manitoba Medical Services. Announcement of the time and place of this open house will be made in the official program.

The Ladies

An attractive program of social events for ladies has been arranged and the wives of members attending are assured of an enjoyable stay in Winnipeg.

Our Exhibitors

With the co-operation of the Medical Exhibitors Association of Canada a full scale display of new and interesting items in pharmacy, equipment, books and food products has been arranged. Members will find that their contacts with representatives of our exhibiting firms will contribute to their store of information.

PROGRAM OF CANADIAN ACADEMY OF ALLERGY, WINNIPEG, JUNE 16

9:30 a.m.—Studies on Urinary Histamine: J. Mitchell, Charles F. Code, and George P. Logan, Mayo Clinic, Rochester, Minn.

10:00 a.m. to 12 Noon: Symposium on Histamine.
(a) Rôles of Histamine in the Anaphylactic Reaction in Guinea Pigs and Rabbits: C. F. Code, Mayo Clinic Foundation, Rochester, Minn.
(b) Liberation of Histamine and Other Substances During Anaphylactic Reactions in Dogs and Monkeys: L. B. Jacques, Saskatoon, Sask.
(c) Rôle of Histamine in Allergic and Anaphylactic Reactions in Man: Bram Rose, Montreal.

2:00 p.m.—Recent Advances in ACTH and Cortisone in Allergy: Bram Rose, Montreal.

2:30 p.m.—Concepts on Mode of Action of Salicylates, L. B. Jacques and J. Lowenthal, University of Saskatchewan.

3:00 p.m.—Experience with Pollen Surveys in Manitoba from 1939 to 1952, C. H. A. Walton and M. G. Dudley.

3:30 p.m.—Recent Advances in Bacterial Allergy: K. A. Baird, Saint John, N.B.

4:00 p.m.—Clinical and Experimental Allergic Cephalalgia: Jacques Leger, Montreal.

4:30 p.m.—Effect of Antihistamines on Smooth Muscle: T. H. Aaron and W. Stewart, Edmonton.

CANADIAN NEUROLOGICAL SOCIETY, ANNUAL MEETING

The Canadian Neurological Society will hold its Fifth Annual Meeting in Winnipeg June 20 and 21.

After registration and meeting of the Council on the first morning the scientific sessions will begin: the speakers in the morning will include Drs. R. T. Ros, H. H. Hyland, D. J. Sirois, E. H. Botterell and J. Auer. In the afternoon Drs. M. S. Saunders, W. Penfield, P. Gloor, H. Jasper and D. B. Tower will present papers. The annual dinner will be held that evening.

The morning of June 21 will be devoted to seven papers, and after lunch there will be a symposium on neuropathies with a panel composed on Drs. R. J. Rossiter on biochemistry; J. W. Scott on Physiology; O. Bailey on pathology and F. L. McNaughton on clinical aspects.

Physicians and others interested in neurology are welcome to attend these sessions.

Ironically enough, pre-employment audiometry is being done only in certain of the non-noisy trades, but in the noisy trades pre-testing is feared both by employers and the unions. Employers are against it because it draws attention to the effect of the job on hearing; unions object to it because pre-employment audiometry might show hearing loss before employment and workers might not be able to collect insurance or compensation later on. He also pointed out that in noisy trades such as shipbuilding, etc., the men, as a rule, do not like to use protective devices such as "ear defenders".—From *The Bulletin*, New York League. (From a lecture by Dr. E. P. Fowler.)

Canad. M. A. J.
June 1953, vol. 68

BY-LAWS CANADIAN MEDICAL ASSOCIATION RECOMMENDED REVISION

WHEREAS by act of the Parliament of Canada dated the nineteenth day of May, one thousand nine hundred and nine, The Canadian Medical Association is empowered to make by-laws and rules as it may deem necessary, and

WHEREAS it has been deemed desirable and expedient that the constitution and by-laws of the said Association be revised, now therefore, BE IT ENACTED that the Constitution and By-Laws be and the same are hereby repealed and the following substituted therefor:

BY-LAWS

(For the government and management of the business and affairs of The Canadian Medical Association.)

CHAPTER I

Title:

This Association shall be known as The Canadian Medical Association, and when the French language is used, it shall be known as "L'Association Medicale Canadienne".

CHAPTER II

Objects:

1. The promotion of health and the prevention of disease.
2. The improvement of medical services however rendered.
3. The maintenance of the integrity and honour of the medical profession.
4. The performance of such other lawful things as are incidental or conducive to the welfare of the public and of the medical and allied professions.

CHAPTER III

The Seal:

The seal which is now in the hands of the General Secretary shall be the seal of The Association.

CHAPTER IV

Ethics:

The Code of Ethics of The Association shall be such as may be adopted by The Association from time to time. A copy shall be supplied to each member of The Association on request.

CHAPTER V

Divisions:

Section 1.

A Provincial Medical Association (or the body representing organized medicine in a Province and enjoying all the rights and privileges of a medical association) may become a Division and enjoy all the rights and privileges of a Division in the following manner:

a. By intimating to the Canadian Medical Association in writing that it desires to become a Division.
b. By agreeing to amend, where necessary, its Constitution and By-Laws to place them in harmony with the Constitution and By-Laws of this Association.
c. By agreeing to collect from those of its members who desire to be members of The Canadian Medical Association such annual fee as may from time to time be set for membership and remit same to this Association.
d. By agreeing to take such steps as seem proper to the Division to increase membership in The Association.

Section 2.

It shall then be known as The Canadian Medical Association (name of Province) Division, but if it choose may retain its pre-existing name as well.

CHAPTER VI

1. *Membership and Discipline:*

The Association shall be composed of ordinary members, members-at-large, senior, non-resident, and honorary members, and they shall be so designated, according to the way in which they qualify under the requirements for classification, which are as follows:

(a) *Ordinary Members:*

Every member in good standing in a Division shall be automatically an ordinary member of The Canadian Medical Association on payment of the annual fee as levied by the General Council.

(b) *Members-at-Large:*

Any graduate in medicine residing in Canada, or any teacher of the ancillary sciences in a school of medicine in Canada (not a graduate in medicine), who is not a member of a Division may be accepted as a member of The Canadian Medical Association provided that, with his application, a certificate of approval from the executive body of the Division in the Province in which the applicant resides be furnished to the General Secretary. In the case of an applicant residing in Canada in a territory beyond the jurisdiction of a Division, the application must be endorsed by two members of The Canadian Medical Association. Such members shall be designated "Members-at-Large" and shall pay the annual fee as levied by the General Council.

This section shall be construed so as to include permanent officers of the Armed Forces, who may be accepted as members without becoming members of a Division.

(c) *Senior Members:*

Any member of The Association in good standing for the immediately preceding ten year period who has attained the age of seventy years is eligible to be nominated for senior membership by an ordinary member of The Association. He shall be approved by the Executive of the Division in which he practised, but he may be elected only by the unanimous approval of the members of the Executive Committee in session present and voting. Not more than eleven such senior members may be elected in any one year. Senior members shall enjoy all the rights and privileges of The Association but shall not be required to pay any annual fee.

(d) *Non-Resident Members:*

Non-resident members may be elected by the Executive Committee from regularly qualified practitioners residing outside of Canada. They shall be required to pay not more than seventy-five per cent of the annual fee as levied by the General Council.

(e) *Honorary Members:*

Honorary members may be nominated by any member of The Association and shall be elected only by a unanimous vote of the Executive Committee or the General Council in session present and voting. Not more than five honorary members may be elected in any one year and at no time shall the list of living honorary members exceed twenty-five. Honorary members shall enjoy all the rights and privileges of The Association but shall not be required to pay any annual fee.

2. *Discipline of Members:*

Any member who fails to conform to these By-Laws and/or the Code of Ethics of this Association shall be liable to censure, suspension or expulsion.

(a) Any member whose annual fee is payable either directly to The Canadian Medical Association or through one of its provincial divisions, and whose annual fee is not paid on or before the 31st day of March of the current year, may, without prejudice to his liability to The Association, be suspended from all privileges of membership.

(b) Any member of The Association who after due enquiry by the Executive Committee shall be judged to have been guilty of disgraceful conduct in any professional respect shall be liable to censure, suspension or expulsion from membership in The Association by resolution of the Executive Committee confirmed by a three-fourths vote at the next Annual Meeting of the General Council.

(c) Should any member of The Association be convicted of any criminal offence, or have his name removed from the register of the Medical Council of Canada, or of the licensing body of any Province of Canada, because of felonious or criminal act, or disgraceful conduct in any professional respect, the Executive Committee may, by resolution, confirmed at the next ensuing annual meeting of the General Council, by a three-fourths vote of those present, censure or suspend or expel such persons from membership in The Association.

(d) Any member suspended or expelled by resolution as aforesaid, shall thereby forfeit all his rights and privileges as a member of The Association.

(e) Any member suspended or expelled by resolution as aforesaid, shall, subject to conditions imposed by the Executive Committee, be restored to membership upon resolution of the Executive Committee confirmed at the next ensuing annual meeting of the General Council.

(f) By accepting membership under the terms of the By-Laws and Code of Ethics and becoming a member of The Association, every member attorns to these By-Laws, and agrees to such right of discipline as aforesaid, and thereby specifically waives any right or claim to damages in the event of his being so disciplined.

(g) *Resignation from Membership:*

Membership in The Association shall automatically cease only on suspension, expulsion or death. Resignation may be effected (1) in the case of a member of a Division by giving notice to the Secretary of the Division not less than one month before the beginning of the calendar year; (2) in the case of a member at large by giving notice directly to the General Secretary of The Canadian Medical Association one month before the next annual fee is due.

(h) *Registration at Meetings:*

No member shall take part in the proceedings of The Canadian Medical Association or in the proceedings of any of the Sections thereof or attend any part of the meeting until he has properly registered. Only members and invited guests are eligible to register and attend an annual meeting.

CHAPTER VII

Affiliated Societies:
Section 1.

Any nationally or internationally organized medical, scientific, or sociological body may, subject to the approval of the General Council, become affiliated with The Canadian Medical Association. Affiliation shall be of two classes:

(a) Canadian Medical Specialist Societies whose members are also members of the Canadian Medical Association.

Canad. M. A. J.
June 1953, vol. 68

These shall, on application to and on approval by the Executive Committee or by the General Council, be accepted as an affiliated body and shall be entitled to one seat on General Council for each society so affiliated. For the purpose of arranging sectional meetings at Annual meetings of the Canadian Medical Association and for such other purposes as the Executive Committee may deem expedient, any such specialist organization in the absence of a corresponding section properly organized and functioning and at the request of the Executive Committee or its properly designated agent may act for and on behalf of the Canadian Medical Association in the performance of those offices and duties which a properly constituted section might otherwise be expected to perform.

(b) Other National or International Associations.

Other national or international bodies as above may become affiliated with the Canadian Medical Association. In particular cases, in which in the judgment of the Council or of the Executive Committee a very close liaison between any such body and the Canadian Medical Association would appear to be likely to advance the interest of Medicine in Canada, then the Executive or General Council may enter into such liaison arrangements as may be found to be mutually agreeable even though this may involve the granting of membership in Council on an exchange basis.

Section 2.

Any affiliation made under this chapter shall mean that a friendly relationship exists between the two bodies. There shall be no obligation on the part of either party to the affiliation, by virtue of such affiliation, to sponsor policies or movements initiated or advanced by or on behalf of the other.

Section 3.

Affiliation shall be on a year to year basis and shall continue without interruption unless either party to the affiliation shall give notice to the other in writing of its intention to withdraw from the affiliation.

Section 4.

Membership in Council shall not generally be a condition of affiliation under Section 1 subsection (b) of this chapter but may be given under special circumstances as therein suggested, and as provided in Chapter XIII Section 1 (k).

CHAPTER VIII

Guests and Visitors:

Section 1. Visitors from Outside of Canada:

Medical practitioners and other men of science residing outside of Canada may attend the annual meeting as guests of the President or of the General Council, or as visitors when vouched for by the General Secretary. They shall register with the General Secretary, without payment of fee and may, after proper introduction, be allowed to participate in discussions.

Section 2. Medical Students Attending Meetings:

Any hospital intern or medical student, when properly vouched for, may be admitted as a guest to the scientific meetings, but shall not be allowed to take part in any of the proceedings unless specially invited by the Committee on Program to present a communication.

Section 3. Delegates from Affiliated Societies at Scientific Meetings:

Two delegates from each affiliated society, one only of whom is required to be a member of this Association, may attend the scientific meetings.

Section 4. Delegates from Affiliated Societies at Meetings of the General Council:

Two delegates from any affiliated society, provided one delegate is a member of this Association, may be invited by the Executive Committee to attend meetings of the General Council. They may, at the request of the Chairman, take part in the deliberations but shall have no voting power.

CHAPTER IX

Meetings:

Section 1. Time and Place of Meetings:

The time and place of meetings shall be decided by the General Council or the Executive Committee, and shall be announced as early as possible.

Section 2. Annual Meetings:

When the Canadian Medical Association meets in a Province, the meeting of the Division of that Province for that year may be for business purposes only. The local arrangements shall be under the direction of the Executive Committee of the Canadian Medical Association, which may enlist the assistance of the Division or one of its component Societies. The Canadian Medical Association assumes full control of the proceedings of the meeting and of all financial obligations save entertainment.

Section 3. Program for Annual Meetings:

The program of the meeting may consist of business sessions, general and sectional sessions, and any other sessions which may be decided upon by the Executive Committee.

Section 4. Presiding Officer:

The President or some person designated by him shall preside at all general meetings.

Section 5. Rules of Order:

The Rules of Order which govern the proceedings of the House of Commons of Canada shall be the guide for conducting all meetings of The Association.

CHAPTER X

Sections:

Section 1. Organization and Functions:

Members of the Canadian Medical Association with the consent and approval of the General Council, may organize a Section for the purpose of: (a) interesting The Canadian Medical Association in a particular field of medicine; (b) voicing considered expressions of opinion for the benefit of The Canadian Medical Association on matters which concern the Section; and (c) arranging for meetings in co-operation with the Central Program Committee.

Section 2. Recognition of Existing Sections:

The following Sections are recognized as existing on June 9, 1952:

- Anæsthesia
- Armed Forces Medical Section
- Dermatology
- General Practice
- Historical Medicine
- Industrial Medicine
- Medicine
- Obstetrics and Gynæcology
- Ophthalmology
- Otolaryngology
- Pædiatrics
- Preventive Medicine
- Psychiatry
- Radiology
- Surgery
- Urology

Section 3. New Sections:

New Sections may be organized on the application in writing of not less than twenty-five members of The Canadian Medical Association setting forth the subject or subjects proposed for study and discussion by the Section and the proposed name of the Section. Such application shall be filed with the General Secretary of The Canadian Medical Association and submitted by him to the next meeting of the Executive Committee after the application is received, and the Executive Committee shall transmit the application to the next meeting of the General Council with its recommendations in respect of the application, and the General Council may grant the application in the form made or with such variations therein as the applicants may approve, or may refuse the application or postpone consideration thereof.

Section 4. Meetings:

The view of our membership being that the unity of The Canadian Medical Association, in all its Sections and Divisions, is essential to the realization of its objectives, it follows that the interests of any Section must rank below those of The Association as a whole and must be required, if necessary, to give place to them in the national interest. Subject to this and by arrangement with the Executive Committee, meetings of Sections shall be held during the time of and in conjunction with the Annual Meeting of The Canadian Medical Association. Other meetings of a Section may be called by the Chairman of the Section with the approval of the Executive Committee. Notice of a meeting of a Section, other than a meeting to be held during the Annual Meeting, shall be given by publication in an issue of the Journal of The Canadian Medical Association published not less than one month prior to the meeting.

Section 5. Officers:

There shall be a Chairman and Secretary of the Section elected at a meeting thereof held during an Annual Meeting of The Canadian Medical Association, and they shall hold office from the close of that meeting until the close of the next meeting of the Section held during an Annual Meeting. In the event of either of the said officers not being elected as aforesaid or resigning or dying or becoming incapacitated during his term of office, the Executive Committee may appoint a member of The Canadian Medical Association to fill the office until the next election.

Section 6. Duties of The Chairman:

The Chairman, or someone designated by him, shall preside at all meetings of the Section, and if he be absent and no one has been designated by him to preside, the meeting of the Section shall elect a Chairman.

Section 7. Duties of The Secretary:

The Secretary of the Section shall keep a correct record of its transactions in duplicate and one copy shall be handed to the General Secretary of The Canadian Medical Association for insertion in the Minute Book provided for the purpose. The other copy shall be retained by the Secretary of the Section for the use of the Section and its officers.

Section 8. Program at Annual Meetings:

It shall be the duty of a Section through its Chairman and Secretary to co-operate with the Central Program Committee, to arrange for the meeting of the Section to be held during the Annual Meeting.

Section 9. Dissolution of Sections:

In the event of it appearing from the small number of registrations in a Section or the failure to hold meetings thereof or on any other ground, that interest in its subject or subjects is lacking, the General Council, on recommendation of the Executive Committee may dissolve the Section, and it shall not be revived except upon a new application for recognition.

Section 10. Authority of the Section:

No Section or meeting of a Section and no officer or officers of a Section shall have the right to speak for The Canadian Medical Association as such, but any resolution passed at a meeting of a Section may, if the meeting so decides, be submitted to the General Council or the Executive Committee of The Canadian Medical Asso-

Canad. M. A. J.
June 1953, vol. 68

ciation for consideration and action, and it shall be the duty of the General Council or the Executive Committee as the case may be to receive such resolution and consider the same and take such action as it may decide in respect thereof at its first meeting after the receipt of such resolution.

CHAPTER XI

Officers, Officials and Executive Committee:
Section 1. Officers and Officials:

The Officers and Officials of The Association shall be:

(a) The Patron.

(b) The Elective Officers, who shall be a President, a President-Elect, a Chairman of the General Council and an Honorary Treasurer.

(c) The appointive Officials who may be a General Secretary and a Deputy General Secretary, an Editor, and such other Officials as may be appointed by the Executive Committee. These appointive Officials shall have no vote at any meetings of The Association nor of any of its Committees.

Section 2. Appointment of Nominating Committee:

(a) The General Council at its first session at the time of the Annual Meeting shall elect by ballot from among its members present a Nominating Committee of TEN, not including the President who shall be ex officio a member of the Committee and the Chairman thereof.

(b) Each Division in The Association is entitled to appoint from amongst its delegates to the General Council one member to the Nominating Committee. Provided this nomination be made in writing to the General Secretary prior to the Annual Meeting and the delegate so nominated be present, he shall be declared elected to membership on the Nominating Committee.

(c) Upon completion of the election of Divisional Representatives as provided for in clause (b) of this section, any vacancies which remain shall be filled by nominations from the floor. Election shall be by majority vote, on a single ballot and the Chairman of the General Council shall if necessary give the casting vote.

Section 3. Duties and Powers of the Nominating Committee:

The Nominating Committee shall meet on the day of its election and submit to a later session of the General Council:

1. Nomination of the following officers of The Association: A President-Elect, a Chairman of the General Council and an Honorary Treasurer.

2. Nomination of an Executive Committee which, in addition to those who are members ex officio (see Chapter XIII, Section 6) shall consist of fourteen members drawn from the General Council and geographically distributed as follows: three shall be resident in each Province in which an office of the Association is located and one shall be resident in each of the other provinces.

3. Nomination from members of the General Council of ten alternates for the elected members of the Executive Committee. There shall be one alternate nominated from each Province. The function of the alternates shall be to act in the place of an elected member of the Executive Committee who is absent because of death or illness or from cause acceptable to the Chairman of the Executive Committee.

4. At its session, the Nominating Committee may receive in writing:

   (a) Each Division's official nomination of the candidate or candidates for representation on the Executive Committee to which the Division is entitled; and also,

   (b) Each Division's official nomination of one alternate who will act in the absence by reason of death or illness or from cause acceptable to the President, of the member or one of the members of the Executive Committee representing that Division. In the event of such an official nomination by a Division being rejected by the Nominating Committee the reasons for such action shall be incorporated in its report to the General Council.

5. *Rules of Procedure:*

   The Committee shall be called to order by the President as Chairman of the Committee. In the absence of the President, the General Secretary shall convene the Committee and request the Committee to select, by open vote, the Chairman. The Committee shall then proceed to carry out its duties by open vote. In case of a tie vote the Chairman shall have the casting vote in addition to the vote to which he is entitled as a member of the Committee. When called for, the report of the Committee shall be presented to the General Council by the General Secretary.

Section 4. Election of Officers and Executive Committee:

When the report of the Nominating Committee has been received by the General Council in session, other nominations may also be received from the floor. A ballot shall then be taken for each of the offices in turn and also for elective membership of the Executive Committee by Provinces.

CHAPTER XII

Duties of Elective Officers and of Appointive Officials:

Section 1. Duties of the President:

The President shall preside at the general session of The Association and shall perform such duties as custom and parliamentary usage require. He shall be required to preside at all social functions of the Association, its Executive or its General Council, or to delegate some other member of the Executive or of the General Council so to do. He shall deliver a presidential address. He shall be a member ex officio of all committees of The Association. He shall be reimbursed for his legitimate travelling expenses while engaged in the business of The Association. He shall be a member ex officio of the Executive Committee for the year immediately succeeding his Presidency.

Section 2. Duties of the President-Elect:

The President-Elect shall be installed and shall assume the office of President at the time of the Annual Meeting next following that of his election to the office of President-Elect. He shall be a member ex officio of all committees of The Association excepting the Nominating Committee. In the event that the office of President of the Association shall become vacant during the term of office of the President-Elect, said President-Elect shall serve also as Acting President and in that capacity shall assume all the powers and duties of the President during the unfinished portion of that presidential term. He shall be reimbursed for his legitimate travelling expenses while engaged in the business of The Association.

Section 3. Duties of the Immediate Past-President:

He shall be a member ex officio of the Executive Committee for the year immediately succeeding the termination of his Presidency and shall be a member of the General Council as provided in Section 1, Sub-Section (g), Chapter XIII of these By-Laws.

Section 4. Duties of the Chairman of the General Council:

The Chairman of the General Council shall preside at all meetings of the General Council. He shall be reimbursed for his legitimate travelling expenses while engaged in the business of The Association. He shall be a member ex officio of all Committees, excepting the Nominating Committee.

Section 5. Duties of the Honorary Treasurer:

The Honorary Treasurer shall be the custodian of all moneys, securities and deeds which are the property of The Association. He shall pay by cheque only. Such cheques shall be signed by two persons authorized by the Executive Committee to sign cheques of The Association and shall be covered by voucher. He shall prepare an annual financial statement audited by a Chartered Accountant. He shall furnish a suitable bond for the faithful discharge of his duties. The cost of the bond shall be borne by the Association. He may receive for his services an honorarium to be determined by the General Council. He shall be reimbursed for his legitimate travelling expenses while engaged in the business of The Association. He shall be a member ex officio of the Executive Committee.

Section 6. Duties of the General Secretary:

The General Secretary shall be the Secretary also of the General Council and of the Executive Committee of The Association. He shall also be a member ex officio of all Committees of The Association. He shall give due notice of the time and place of all annual and special general meetings, by publishing the same in the official Journal of The Association, or, if necessary, by notice to each member. He shall keep the minutes of the meetings of the General Council and of the Executive Committee in separate books and shall provide minute books for the secretaries of the different sections which he shall require to be properly attested by the secretaries thereof. He shall notify the officers and members of committees of their appointment and of their duties in connection therewith. He shall publish the official program of each annual meeting. He shall perform such other duties as may be required of him by the President, the General Council or the Executive Committee. All his legitimate travelling expenses shall be paid for him out of the funds of The Association and he shall receive for his services a salary to be determined by the Executive Committee.

Section 7. Duties of the Deputy General Secretary:

The Deputy General Secretary shall be the chief Assistant Secretary and as with any other assistant secretaries shall perform such duties as shall be assigned by the Executive Committee or by the General Secretary. On special assignments and in the absence of the General Secretary he shall act in the interest of The Association with all the obligations and authority of the General Secretary as provided in Section 6 of this Chapter. All his legitimate travelling expenses shall be paid for him out of the funds of The Association and he shall receive for his services a salary to be determined by the Executive Committee.

Section 8. The Editor of the Journal:

The Editor of the Journal shall be responsible to the Executive Committee for the regular production of the Journal of The Association, and

Canad. M. A. J.
June 1953, vol. 68

within the usually recognized limits, for its scientific and literary standards of quality. Having respect to the general policy of The Association he shall publish such information and editorial comment as the time and circumstances may require and as may be to the interest of Canadian Medicine.

He shall be expected to attend meetings of the Executive and of General Council and to perform such duties as may properly be expected of his office and as may reasonably be required by General Council or by the Executive Committee. All his legitimate travelling expenses shall be paid for him out of the funds of the Association and he shall receive for his services a salary to be determined by the Executive Committee.

CHAPTER XIII

The General Council:

Section 1. Organization:

The General Council shall consist of:

(a) The members of the Executive Committee.
(b) The Officers and Officials of The Association.
(c) The Presidents and Secretaries of Divisions.
(d) The Divisional Delegates, which shall include the nominees to the Executive Committee and the Nominating Committee.
(e) The Chairman of Standing Committees.
(f) The Chairmen of Organized and Recognized Sections.
(g) The Past Presidents of The Association.
(h) The Deputy Minister of National Health.
(i) The Director General of Medical Services of the Department of Veterans' Affairs.
(j) A representative of The Association of Canadian Medical Colleges who is a Dean and a Member of this Association.
(k) Representatives of Affiliated bodies as in Chapter VII, Section 1 (a) and (b) and Section 4.

Section 2. Election of Delegates:

Each Division shall be entitled to elect five delegates to serve on the General Council for its membership in The Canadian Medical Association of fifty or less; one additional delegate for its membership from fifty-one to one hundred; one additional delegate for its membership from 101 to 300; and thereafter one delegate for every 300 above 300. One of its representatives on the General Council may be named by a Division as its nominee to the Nominating Committee of The Association.

Section 3. Meetings of the General Council:

The General Council shall meet for at least the first two days of the Annual Meeting of The Association and thereafter, while The Association is in session, at the call of the Chairman. Before the close of the Annual Meeting it shall elect the officers and the Executive Committee and select the place for the next annual meeting, or, if thought advisable, for meetings up to three years in advance.

Section 4. Special Meetings of the General Council:

During the interval between annual meetings the General Council shall meet at the call of the Executive Committee. For all such meetings of the General Council due notice shall be sent to each member, stating the purpose of the meeting. The Executive Committee, if it so decide, instead of calling such meetings of the General Council may refer important questions to the General Council and obtain its decision by means of a mail ballot. In the event of a mail ballot being taken, two-thirds majority vote shall govern.

Section 5. Duties and Powers of the General Council:

1. The General Council shall act for the Association in all matters not otherwise reserved and more specifically shall as far as possible deal with and dispose of all matters relating to:
   (1) The reports of the Executive Committee and the reports of all Standing Committees and all Special Committees of the General Council.
   (b) Any business originating in or relating to the Divisions which is for the general welfare of the public, the profession or The Association.
   (c) Business which may result from petitions, appeals, recommendations or complaints.
   (d) The election of the officers of The Association, the Nominating Committee and the Executive Committee.
2. It may make By-Laws and regulations and revise them from time to time and it may perform such other acts not elsewhere excluded as shall make for the welfare, order and good government of this Association. Any By-law or regulation so made or any revision thereof shall become effective when adopted by a majority of the members present and voting at any annual or special General Meeting of the Association as provided in Chap. XVII Section 3 of these By-laws.
3. It shall have supervision of all properties and of all financial affairs of The Association. It shall keep a record of all meetings and of the receipts and expenditure of all funds, and shall report upon same in the Journal after the Annual Meeting.

Section 6. The Executive Committee May Act for the General Council:

In order that the business of The Association may be facilitated during the interval between meetings of the General Council, the Executive Committee shall meet from time to time at the call of its Chairman, and shall have all the rights

and powers of the General Council. It shall conduct all necessary business. In case of a vacancy in any office on account of death or otherwise, it shall have power to appoint a successor. In case of a vacancy occurring in the Executive Committee itself by death or otherwise, it shall have power to appoint a successor upon receiving an official nomination from the Division concerned.

The President, the President-Elect, the immediate Past-President, the Chairman of the General Council, the Honorary Treasurer, the General Secretary, the Deputy General Secretary, the Editor and the Managing Editor shall be members ex officio of the Executive Committee, but only the elective officers shall have the right to vote.

CHAPTER XIV

Committees:

Section 1. The Committees of The Association shall be:

(a) Statutory Committees.
(b) Standing Committees.
(c) Special Committees.

Section 2. Appointment of Committees:

(a) Statutory Committees shall be:
The Nominating Committee.
The Executive Committee.
Both of which shall be elected by the General Council.

(b) Standing Committees:
The Executive Committee shall have power to establish Standing Committees, to vary their number from time to time and to discontinue their activities. The Chairmen of Committees, designated by the Executive Committee as Standing Committees, shall be appointed by the Executive Committee which in addition to the duties provided in Chapter XI, Section 3 of these By-Laws, shall also provide or vary their terms of reference. These shall report to the General Council after submitting copies of their report to the Executive Committee as required.

Subject to the reservations contained in this Section, the list of Standing Committees shall now be:

1. The Committee on Cancer.
2. The Committee on By-Laws.
3. The Committee on Economics.
4. The Committee on Hospital Service and Accreditation.
5. The Committee on Legislation.
6. The Committee on Medical Education.
7. The Committee on Pharmacy.
8. The Committee (Central) on Programs.
9. The Committee on Public Health.
10. The Committee on Public Relations.
(It is recommended that the following be included under Public Health as Sub-Committees:
(i) Maternal Welfare.
(ii) Industrial Medicine.
(iii) Mental Hygiene.
(iv) Nutrition.)

(c) Special Committees:
Special Committees may be appointed by:
1. The President.
2. The General Council.
3. The Executive Committee.
4. The Chairman of the General Council.

A Special Committee shall be a short-term Committee and shall assume by direction such duties as are allotted to it. It shall make progress reports to the Executive Committee at each of the meetings of that body, and at any other time that such reports may be required. If its work is likely to be continued, it shall become a Standing Committee on being so designated by the Executive Committee.

Section 3. Duties and Powers of the Executive Committee:

The Executive Committee shall hold one or more sessions before the close of the Annual Meeting at which it is elected. At its first meeting it shall elect its Chairman and appoint the Chairmen of the Standing Committees for the ensuing year. Between the meetings of the General Council, the Executive Committee shall represent the General Council in all its business affairs and shall exercise all the rights and powers of the General Council. The Executive Committee shall report to the General Council at the Annual Meeting and at such other times as the Chairman of the General Council may request.

The Executive Committee may meet when and where it may determine. On the request in writing of any three members (with voting power) of the Executive Committee, the Chairman shall call a special meeting. Seven members (with voting power), exclusive of the Chairman, shall constitute a quorum for the transaction of business.

The Chairman of the Executive Committee instead of calling a meeting thereof *may* and, if requested so to do in writing by any three members of the Committee, *shall* take a mail ballot of the elective members of the Executive Committee on any urgent matter and an affirmative vote by two-thirds of such members shall have the same force and effect as a resolution duly passed at a regular meeting of the Executive committee, provided such mail ballot is taken in the following manner:

The question submitted shall be in a form to which an affirmative or negative answer may be

Canad. M. A. J.
June 1953, vol. 68

given. The ballot shall be sent by prepaid registered post to all elective members of the Executive Committee not less than ten days before the last return date, accompanied by a letter signed by the Chairman of the Executive Committee setting out the circumstances of the emergency and giving the last date on which ballots will be received and requesting that ballots be signed and returned to the Secretary of The Association by such elective members by the date named. Simultaneously with the sending out of the ballots to the elective members of the committee, a copy of the aforesaid letter shall be mailed to those members of the Executive Committee who are not entitled to vote, together with a copy of the question which is being submitted to the elective members. No ballot will be counted unless it is signed by an elective member of the Executive Committee and is in the hands of the Secretary of The Association not later than the return date named. Each elective member may cast one ballot only.

The Executive Committee shall be responsible for the appointment of the appointive officials, shall designate their responsibilities and fix their salaries.

The Executive Committee shall have charge of the publication of the official Journal of The Association and of all published proceedings, transactions, memoirs, essays, papers and programs of The Association.

The Editor and Managing Editor shall present annual reports to the General Council and interim reports at each meeting of the Executive Committee. The Editor shall be reimbursed for his legitimate travelling expenses incurred on Association business. The Executive Committee may appoint Editorial Boards to assist the Editors.

The Executive Committee shall appoint the Auditor and shall have the accounts of the Honorary Treasurer audited annually, or more often if desirable, and shall make an annual report on the same to the General Council.

Each member of the Executive Committee shall be reimbursed for his legitimate travelling expenses incurred in attending meetings of the Executive Committee other than the first meeting or meetings of the new Executive Committee, which may be held before the close of the annual meeting.

Section 4. Duties of Standing Committees:

(a) *Committee on Cancer:*

This Committee shall act in an advisory capacity on all matters relating to the study and control of cancer.

(b) *Committee on By-Laws:*

To the Committee on By-Laws shall be referred all matters relating to the subject before action thereon is taken by the General Council.

(c) *Committee on Economics:*

It shall be the duty of the Committee on Economics excepting where otherwise provided, to deal with (a) social legislation which includes medical services or benefits presumably for medical services; (b) remuneration and employment of physicians by lay bodies, hospital or official bodies, including Federal, Provincial and Municipal Governments; (c) to report thereon with such recommendations as it may see fit to the General Council.

(d) *Committee on Hospital Service and Accreditation:*

(NOTE: Because of a changing relationship with hospitals the final picture of which cannot be projected, it is recommended that the duties of this Committee shall be as may from time to time be determined by the General Council or by the Executive Committee.)

(e) *Committee on Legislation:*

This Committee shall be responsible for following legislative trends and impending specific acts which in any Division or at the National level may be considered as affecting the health of the nation or in any other way as being of concern to the Canadian Medical Association. It shall have as corresponding members the chairmen of similar committees set up by the Divisions of the Association. It shall keep the Executive Committee apprised of such trends or impending acts as it may regard as significant. Matters requiring legislative action arising within The Association may be referred by the Executive Committee to this Committee for consideration and advice.

(f) *Committee on Medical Education:*

To the Committee on Medical Education shall be referred all matters pertaining to medical colleges and medical education. It shall report upon the condition of medical education throughout Canada and upon any proposed change, and may suggest methods for the improvement of medical education.

(g) *Committee on Pharmacy:*

It shall be the duty of the Committee on Pharmacy to deal with (a) all matters arising out of the British Pharmacopœia or any Canadian Formulary or Pharmacopœia; (b) all matters arising out of the drug section of the Food and Drugs Act, the Narcotic Act, or the Patent and Proprietary Medicine Act; and (c) any inquiries from members of The Association relating to the use or standards of drugs.

(h) *Committee (Central) on Programs:*

This Committee, with the assistance of the Chairman and Secretary of each section, shall

have complete charge of the preparation of the scientific program for the Annual Meeting.

(NOTE: It is the recommendation of this Committee on By-Laws that in any year in which a Section is represented in the program the representative of that section shall be an integral part of the Central Program Committee.)

(i) *Committee on Public Health:*

It shall be the duty of this Committee to consider and report upon such matters in the realm of Public Health as should properly engage the attention of The Association and as may be approved by the Executive Committee.

Section 5. Reports of Committees:

Reports of all Committees shall be printed and mailed to all members of the General Council at least one week before the annual meeting.

Section 6. Limitation of Committees re Finances:

No Committee shall expend any moneys or incur any indebtedness or obligation on behalf of The Association without the sanction of the General Council or the Executive Committee.

CHAPTER XV

Addresses and Papers:

Section 1. Addresses at Annual Meeting:

All addresses delivered at an annual meeting shall immediately become the property of The Association, to be published or not, in whole or in part, as deemed advisable, in the Journal of The Association. Any other arrangement for their publication must have the consent of the author or of the reader of the same and of the Editor of the Journal.

Section 2. Publication of Papers Presented at Annual Meeting:

All papers, essays, photographs, diagrams, etc., presented in any Section shall become the property of The Association to be published in the Journal of The Association or not, as determined by the Editor, and they shall not be otherwise published except with the consent of the author and of the Editor of the Journal.

Section 3. Disposition of Papers Presented at Annual Meeting:

Each author of a paper read before any Section shall, as soon as it has been read, hand it with any accompanying diagrams, photographs, etc., to the Secretary of the Section before which it has been presented. The Secretary shall endorse thereon the fact that it has been read in that Section, and shall then transmit it to the Editor of the Journal.

CHAPTER XVI

The Office:

Until changed by the General Council, the offices of The Association shall be at Toronto and Montreal.

CHAPTER XVII

Amendments:

Section 1:

Notice of motion by one or more members to amend the By-Laws, must be placed in the hands of the General Secretary three months before the date of the annual meeting.

Section 2:

Amendments may be proposed by the General Council, the Executive Committee, or the Committee on By-Laws without notice of motion but the proposed amendments shall be published in the Journal at least two months preceding the annual meeting, or by special communication to each member of the General Council four weeks before the annual meeting.

Section 3:

These By-Laws may be amended by a two-thirds vote of the members of the General Council in session present and voting and by a majority vote of a duly advertised General Meeting of the members of The Association.

NOTE: Throughout these By-Laws, masculine designations are to be interpreted as including feminine.

MEDICAL SOCIETIES

SASKATCHEWAN SURGICAL SOCIETY

The fifth annual meeting of the Saskatchewan Surgical Society was held in Regina on April 11 and 12 in the Regina City Public Health Building. These greatly appreciated facilities had been made available through the courtesy of Regina's amiable and able Medical Health Officer, Dr. Walton.

During the first afternoon, under the chairmanship of Dr. D. C. MacRae, four excellent papers were given. Dr. G. M. Malone discussed "Melæna"; Dr. J. A. Brown, "Vaginal Hysterectomy"; Dr. J. H. Wilfong, "Megacolon" and Dr. W. G. Bigelow of Toronto discussed "Research Problems in Cardiac Surgery", and illustrated his talk with a coloured film showing some recent work done at the Banting Institute in Toronto.

On April 12, under the Chairmanship of Dr. A. C. Taylor, Dr. H. J. Spooner and Dr. A. J. Longmore discussed four cases of Tetanus and the following papers were presented: "Peripheral Vascular Disease" by Dr. Bigelow, "Thoracic Surgery in Tuberculosis" by Dr. Harold Anderson and "Facial Injuries" by Dr. J. Szlazak.

In the afternoon following the question and answer period, the following Directors for the ensuing year were appointed: Dr. F. E. Waite, Dr. J. E. Leddy, Dr. D. C. MacRae, Dr. A. L. Caldwell, Dr. J. A. Brown, Dr. C. J. Houston, Dr. C. H. Crosby, Dr. A. C. Taylor, and Dr. F. H. Wigmore. This Board then appointed Dr. Waite as President, Dr. A. C. Taylor as Vice-President, and Dr. J. E. Leddy as Secretary-Treasurer.

Next year's meeting will be held in Saskatoon.

SAINT JOHN MEDICAL SOCIETY

The Saint John Medical Society presented a Spring Clinical Session on April 22, 23 and 24 at the Admiral Beatty Hotel and the Saint John General Hospital.

Dr. R. F. Farquharson, Professor of Medicine of Toronto University was welcomed as our special guest speaker. He was introduced at a dinner in the evening of April 22. Dr. J. A. Finley, President of the local Society presided at the dinner and at the following Clinical Meeting, at which Dr. Farquharson spoke on "Disorders of the Biliary Tract". Discussions were led by Dr. H. H. MacKinnon of Fredericton and Dr. G. F. Skinner. On Thursday, Dr. Farquharson's paper on "Treatment of the Aging Patient" was discussed by Dr. A. B. Walter and another address on "ACTH and Cortisone; Their Value in Therapy" was followed by discussions by Dr. H. O. Tonning and Dr. R. T. Hayes.

Friday, a paper by the guest speaker on "The Menopausal Patient" was discussed by Dr. G. M. White. A Ward Clinic on Diabetes by Dr. Robert Washburn was exceptionally well received.

Papers by N.B. Specialists were: (1) "Contact Dermatitis"—Dr. J. G. MacLean. (2) "Differential Diagnosis of Arthritis"—Dr. H. O. Tonning. (3) "Poliomyelitis"—Dr. A. F. Torrie, Fredericton. (4) "Diagnosis and Treatment of the Neurogenic Bladder"—Dr. H. L. Rosen.

Panel Discussions.—(1) "Surgical Conditions of the Upper Abdomen"—Dr. G. F. Skinner, Dr. J. A. Finley, Dr. G. E. Chalmers, Dr. D. A. Thompson and Dr. I. A. MacLennan. (2) "Investigation of Anæmia"—Drs. A. L. Donovan, C. L. Dumont, H. H. MacKinnon, H. O. Tonning, F. H. George and E. A. Branch. (3) A Clinical Pathological Conference by Dr. H. H. MacKinnon and Dr. H. A. Bird.

Aside from these features, a number of demonstrations and discussions were presented by department groups. Chairmen at the various sessions were Dr. J. A. Finley, Dr. W. D. Miller, Dr. H. A. Bird, Dr. A. L. Donovan and Dr. R. A. H. MacKeen. The attendance throughout the three days was most encouraging and this writer, as an imbiased bystanding observer, was pleased to hear very favourable comments from our out-of-town physician visitors. Slightly better than one hundred were registered including two visitors from the State of Maine. The committee in charge of programs and arrangements comprised: Dr. H. A. Bird, Chairman; Dr. A. L. Donovan; Dr. W. D. Miller and Dr. F. L. Whitehead.

SPECIAL CORRESPONDENCE

The London Letter

(From our own correspondent)

Reviewing the Cost of Health

With the exception of the more vocal section of the Labour Party, there has been a widespread welcome for the announcement of the Minister of Health that he is proposing to set up, under the chairmanship of a distinguished Cambridge economist, a small independent committee to investigate the National Health Service, with a view to "suggesting means, whether by modifications in organization or otherwise, of ensuring the most effective control and efficient use of such Exchequer funds as may be made available" and "how, in view of the burdens on the Exchequer, a rising charge upon it can be avoided, while providing for the maintenance of an adequate service".

This review is just five years late, but it says much for the initiative and understanding of Mr. Iain Macleod, the present Minister of Health, that he should have taken a step which all his predecessors in office have lacked the political courage to sponsor. What has been consistently lacking in the evolution of the Service has been any long-term policy, balancing the various components of the Service and correlating the over-all expenditure with what the nation can afford to spend on the Service. This new, independent, and non-political committee is precisely what is required to investigate the problem and all who are concerned with the physical wellbeing of the nation are convinced that at long last we may obtain what in popular parlance might be called a blue-print for the organization of a National Health Service which will give value for money and at a cost not exceeding what the nation can afford to pay.

B.M.A. and TV

As an appendix to its annual report for 1952-53, the Council of the British Medical Association publishes a report on "indirect methods of advertising". The most topical part of this is the section dealing with broadcasting and television. "The Association accepts the view . . . that medical practitioners who possess the necessary talent and knowledge should be permitted to participate in radio and television programs, provided they ensure the observance of appropriate ethical safeguards". Last year the Representative Body of the Association adopted a resolution to the effect that "practitioners approached to appear in such programs, whether for 'sound' or 'visual' broadcasting, should insist on anonymity as part of the contract". The amplification of this, in the present report, is now quoted *in toto*, and with only one comment —that it would be an interesting psychological (and ethical?) study to discover how the second sentence would be interpreted in practice in different parts of the world.

"Anonymity may be defined for this purpose as withholding the publication or announcement of the practitioner's name, or any information, such as particulars of the appointments he holds, which might enable the public to identify him. Identification cannot be completely excluded, but it is incumbent upon medical practitioners who take part in sound or television programs to ensure that the possibility of identification and publicity is reduced to a minimum. Unless a practitioner insists on anonymity he is not only offending against the ethical principles of the profession but is placing himself in danger of being accused of violating the Warning Notice of the General Medical Council. Should a practitioner, while carefully observing these precautions, be recognized by a section of the public, that fact should not of itself be regarded as evidence of an infringement of the rule of anonymity."

Blood Transfusion

One of the many interesting sections in the recently published Part III of the Ministry of Health's Report for April 1950-December 1951, entitled "On the State of the Public Health", is that dealing with blood transfusion. The donor panel increased from 428,394 in 1950 to 465,137 in 1951. The over-all rejection rate of donors was 2.6%. During 1951, 505,007 bottles (each containing 420 ml.) of blood were issued, compared with 446,390 in 1950. In addition, the Ministry bought and distributed 10,000 bottles of dextran solution. There were 21 cases of homologous serum jaundice, of which three proved fatal. Attention is drawn to the fact that the amount of blood issued during 1951 was about three times greater than in 1946, and the comment is made: "Although little of this blood was wasted in the more usual sense of the word, some was undoubtedly wasted in the sense that it was given to patients in whom insufficient indication for transfusion existed. . . . The institution of some form of control over the use of blood will become necessary if the demands increase at the present rate".

Improved Physique of Children

The annual report on the health of the schoolchildren of Glasgow shows an encouraging improvement in physique. Of the pupils examined, 42.8% had no defect of any kind—the highest proportion since the institution of routine medical inspection of school children in Glasgow in 1909. Except for the 5-year-olds, average measurements all showed an improvement. Thus, com-

paring 1952 with 1945, in the 9-year-old group the average measurements for height rose by 0.56 inch in boys and 0.42 inch in girls, whilst the corresponding increases in weight were 1.02 lb. and 1.08 lb. In the 13-year-old group the corresponding increases were 0.62 inch and 1.65 lb. for boys and 0.32 inch and 1.75 lb. for girls.

WILLIAM A. R. THOMSON

London, May, 1953.

OBITUARIES

DR. ALAN BEECH of Port Washington, B.C., in the Gulf Islands died recently. Dr. Beech was 71, and thouugh he had nominally retired some years ago, had never stopped working because he felt the people of Port Washington needed a doctor to look after them. Alan Beech was the son of Col. Lionel Beech, M.D., who was the first doctor on Salt Spring Island. He was the first intern at the Vancouver General Hospital. He served as major in both world wars, and practised for sixteen years at Salmon Arm between wars. Alan Beech was a very fine man. His personality was charming, though he had a rather shy and retiring manner. Modest and self-effacing, he had a very high sense of duty, and never spared himself. He had a host of friends.

J. FREDERICK BURGESS

AN APPRECIATION

J. Frederick Burgess gained international distinction in Dermatology but his greatest contribution was to Canadian dermatology. He was a charter member of the Montreal Dermatological Society and was instrumental in founding and fostering this Society to its present heights. He took an active part in the founding of the Canadian Dermatological Association and in the establishment of the requirements for Certification and Fellowship in Dermatology in the Royal College of Physicians and Surgeons of Canada. Dr. Burgess was always interested in teaching and will be remembered by hundreds of McGill graduates. In the years following the close of the second Great War he became interested in postgraduate teaching and attracted several young men to his clinic for this purpose. While his health did not permit him to take too active a part in this project, it was he who developed a group of competent associates to carry on this work. The Department of Dermatology at The Montreal General Hospital grew in size and stature under his Chairmanship and remains as a monument to his administrative ability.

Always a keen investigator, he stimulated others to investigative work, not by direction but by example. He was always on the alert for a solution to the many unsolved dermatological problems and was actively engaged in these pursuits when his untimely death intervened.

As one who was privileged to know him and work with him, only in the past decade, I wish on behalf of the postgraduates who were fortunate to train under him, to pay him tribute. "The Chief" will be sorely missed by all of us and by Canadian Dermatology.

R. ROY FORSEY

AN APPRECIATION

Not very often do men overcome the handicap of losing an arm to the extent that Fred Burgess did. He must have wondered when he got back from the war just what he would do. But once he began he showed no hesitation at all in going ahead with driving ceaseless energy. He was one of the younger men who came on to the Montreal General staff at a time when research in that institution was expanding rapidly. Fred took up the study of skin disease associated with fungi and soon made himself known in this field. He found an ideal mentor in the late Dr. Laurence Rhea, in whose laboratory the culture work was done, and whose genius for detecting merit and encouraging effort was unexcelled. In one of Rhea's annual hospital reports he said of Fred's work: "The very satisfactory results he has obtained have not come as a result of accident but by persistence, energy, good judgment and careful analysis of his mistakes".

To these qualities might be added, and Rhea would have been the first to agree, a pleasing personality. Student as he was Fred had a wide circle of friends both in his work and recreation. His charming photographic studies of fungi showed an artistic sense of high order, and he had the rare capacity of being able to give at a medical society meeting an illustrated talk on molds which was as satisfying to the eye as to the mind.

In his later years he suffered much illness, and yet he was eager and interested in his work to the last. Somehow he managed to avoid the outward manifestations of growing older. He leaves the memory of a good friend and of a keenly inquiring mind.

H. E. M.

DR. LEO M. COUGHLIN of Vancouver, B.C., died suddenly on March 18 in Shaughnessy Hospital. He was 60. A graduate of the University of Toronto, he had practiced here for the last 12 years. Dr. Coughlin served in both world wars and won the rank of major in the second. Surviving is his widow.

DR. JEAN DELAGE of Montreal, died on April 5 after a long illness. Born in Ste. Madeleine, he studied at the St. Hyacinthe Seminary and completed his medical studies at the University of Montreal. He was named Rougier Fellowship award winner in anatomy and did postgraduate work in Paris. On his return to Canada, he began teaching anatomy at the University of Montreal, and, in 1949, after 23 years teaching medical science to hundreds of students, he retired due to poor health. At the time of his death he was with the health department of the City of Montreal. He is survived by his widow and a son.

DR. J. A. STEWART DORRANCE, son of Professor and Mrs. R. L. Dorrance, Queen's University, died suddenly at his parents' home on April 21. Dr. Dorrance graduated from Queen's University in 1947. After interning in the Kingston General Hospital, he joined the staff of the Children's Memorial Hospital, Montreal. For the past year he has been conducting investigational work under Drs. Ford Connell and D. L. Wilson of the Department of Medicine, Queen's University.

AN APPRECIATION

The sense of loss by death is always heightened for those who die young. Stewart Dorrance gave great promise. Even with the grave handicap of chronic disease he was sanguine and mentally active. He was of great assistance in supplying our Journal with abstract material, and he will be missed, not only for his help, but for his eager mind and pleasant nature.

H.E.M.

DR. HUGH A. FARRIS died in the Saint John General Hospital on April 25, 1953 after a two day illness. Born in 1881 at White's Cove Queens County, Dr. Farris' early education was received at his birthplace, Gagetown Grammar School, Fredericton High School and Acadia University. He graduated from McGill Medical School in 1907.

He was director of the Saint John Tuberculosis Hospital from 1915 to 1930. Following this, he carried on a practice confined to heart and chest diseases. Dr. Farris was a Fellow of the Royal College of Physicians of Canada, a Fellow of the American College of Physicians and a member of the American Clinical Medicine and Clinalological Society, the Canadian Medical Association, The Canadian and American Tuberculosis Associations, The New Brunswick Medical Society and the American Pathological Society. He was a Baptist. He is survived by three brothers.

Canad. M. A. J.
June 1953, vol. 68

AN APPRECIATION

Hugh Farris was a man of enthusiasm, not always practical but always expressed in forceful words without fear or favour. His friendship was a tricky possession. One was always uncertain as to where one stood depending on agreement or disagreement with any current enthusiasm, but in any case of disaster or hard luck, when the chips were down, Hugh could be depended upon to support wholeheartedly anyone who had received the friendship which he sometimes made difficult. In his active medical life, this fire of spirit burned brightly in his life-long interest in curing and preventing tuberculosis. The mark of his devotion is written into the fine structure of the tuberculosis care now evident in his loved native Province of New Brunswick.

When his physical forces were somewhat spent and his practice restricted to old friends, he took great interest in music festivals in New Brunswick and also in amateur painting of the local scene, which continued to excite him until his sudden death. He was a life long Liberal and his belief in his party was that of almost fanatical sincerity which makes a great party greater. He was a brilliant son of a proud family whose roots were and are deep in Maritime tradition. He had many friends and it cannot be said he had no enemies for his stand on any question was always so forthright that there was little room for compromise, so that universal popularity was impossible. This can be said, his province and Canada are better for his contributions to the life of the nation and to the lives of its citizens.

A. S. KIRKLAND

DR. GEORGE DAVY GORDON of Kemptville, Ont., died on March 31. He was 72. He had practiced for the past 38 years and was active until he suffered a stroke two weeks ago. Previous to coming to Kemptville, Dr. Gordon practiced for six years at the Village of Bishops Mills. Born in Kingston, he took his medical training at Queen's University, graduating in 1906. During the First World War, Dr. Gordon served as a major in the R.C.A.M.C. In the Second World War, he served as medical officer at the Kemptville Officers' Training School. Surviving in addition to his widow is a son, Dr. William J. Gordon of Peterborough.

DR. THOMAS JAMES GRAY, of Saskatoon, died in a local hospital on March 8 at the age of 72. Dr. Gray was born in Kingston, Ontario, and graduated from Queen's University in 1905. He practiced in Portage la Prairie, Man., for a short time before he went to Humboldt where he stayed until his enlistment in the army in 1914. He served overseas from 1914 until 1918. He returned to take charge of the military hospital in Moose Jaw until it was closed in 1920. He took up practice in Saskatoon in 1922 and continued until his retirement to Vancouver in 1942. He returned to Saskatoon last August. He is survived by his widow.

DR. FRANCIS T. GREEN, of Stoney Creek, Ont., died at the Hamilton General Hospital on March 16. Eighty-four years of age, he was, at the time of his death, the oldest living descendant of the first settler in the Stoney Creek district. He was born in Saltfleet Township. His grandfather, Samuel Kennedy Green, and his great-grandfather, Samuel Green, the first settler in the area, came from the state of New Jersey to settle in that part of the country known later as the Village of Stoney Creek, where, in June, 1791, he staked out his claim.

Dr. Green received his early education at Stoney Creek and graduated in medicine in 1891. He practised at Wellandport from 1892 to 1900, when he returned to Stoney Creek. There, he was the "family doctor" until he retired from active practice 41 years later. Always a lover of horses, he became an ardent harness racing enthusiast following his retirement and owned several racing horses. He leaves his widow, two sons, Dr. Francis T. Green, of Burke's Falls, Ontario, and Dr. Lloyd S. Green of Hamilton, and four daughters.

DR. G. LYALL HODGINS, O.B.E., aged 63, former chief of medical staff at Vancouver General Hospital, died on April 2 in Phoenix, Arizona. Born at Lucan, Ont., Dr. Hodgins graduated from University of Toronto in 1911. He interned at Norwegian-American Hospital, Chicago, after which he was certified as a specialist in internal medicine with the R.C.P.S.[C.]. Before coming to Vancouver in 1920, Dr. Hodgins practiced six years in Toronto and a year in Regina.

In 1926 he went into practice here and later pioneered in the medical clinic field in western Canada, establishing Vancouver's first clinic. Dr. Hodgins was named chief of medical staff at Vancouver General Hospital in 1946 and resigned this post late in 1950. During his association with Vancouver General, Dr. Hodgins took a keen interest in occupational therapy and the Women's Auxiliary and is credited with much of their advancement. He was on the council of the Royal College of Physicians and Surgeons of Canada for a number of years, a member of the C.M.A. Besides his widow, Dr. Hodgins is survived by a son and a daughter.

DR. ALLAN N. KITT, of Sprucedale, Ont., formerly of Lucan, died recently in St. Joseph's Hospital, Parry Sound. Born in Lucan, he graduated from Lucan High School, and then went to Victoria College, where he took his B.A. degree in 1909. He graduated from University of Toronto Medical School in 1911, and then practised at Byng Inlet for a short time. In 1914 he went to Whitney, and was there until October 1922, when he began a practise in Sprucedale. In 1943 he assumed temporary charge of Dr. Malkin's office in Parry Sound, remaining there until he retired to Sprucedale in 1947, where he resided until his illness. He had been an active member of the school board and of various other organizations. Surviving is his widow.

DR. B. E. LANG, aged 73, died on February 27 in St. Paul's Hospital, Vancouver, B.C. after a lengthy illness. He was born at Exeter, Ont., and came to Vancouver in 1911 after taking his degree in medicine at University of Manitoba and doing postgraduate work in London, England. During World War One, Dr. Lang served overseas with the Imperial forces as a captain in the Medical Corps. An ardent huntsman and fisherman, he was active in sports circles here for many years. Dr. Lang is survived by his brothers, Dr. W. H. Lang of Vancouver and Rev. David Lang, D.D., former minister of St. Andrew's Presbyterian Church, St. John's, Nfld.

DR. CHARLES A. MACKENZIE, died April 6 at the age of 83 and the flag over the Medical Arts Building in Winnipeg flew at half-mast from April 6 to April 9 as a mark of respect. For twenty-five years he was president of the building, erected for and owned by doctors and dentists. With the late Dr. Harvey Smith he inspired the plan of erecting an office building for the profession and when it was constructed, administered its finances with marked ability.

Born in Ontario, he was educated at Upper Canada College and received his medical training at McGill, graduating in 1899. Coming to Winnipeg he served for many years as medical officer for the Canadian Northern railway, of which his uncle Sir William Mackenzie was one of the founders. In connection with the visit of the British Medical Association to Winnipeg in 1930 he acted as treasurer of the group of Manitoba doctors set up to arrange for the meeting. He was President of the Manitoba Medical Association in that year. He is survived by his widow, and two daughters.

DR. JAMES M. MORROW died in the Fisher River Hospital, Hodgson, Man. on April 14. Born in Lanarkshire, Scotland, he came to Winnipeg in 1913 and graduated from Manitoba Medical College in 1919. After practising at Yorkton and Winnipeg, he joined the Indian Health Service in 1921 and served in Saskatchewan, British Columbia and Manitoba. He is survived by his widow, a daughter and two sons, one of whom, Dr. James J. Morrow, practises at Sherridon, Man.

DR. G. I. NUGENT of Fredericton died in St. Petersburg, Florida on April 16. Dr. Nugent was born in Chipman, N.B. in 1883. His primary education was at Chipman and at Fredericton High School. Later, he received his B.A. from the University of Kansas and graduated in Medicine from the University of Oklahoma. Intern training at New York Polyclinic Hospital was followed by general practice at Chipman for some years before moving to Fredericton. Dr. Nugent was a member of the Fredericton Medical Society, The N.B. Medical Society, The Canadian Medical Association and several Masonic organizations. Dr. Nugent was deeply interested in all his fraternal relations and had a real gift of friendliness. He is survived by his widow and an adopted daughter.

DR. MANLEY G. PEEVER, of London, Ont. died quite suddenly on March 17 at St. Joseph's Hospital. Dr. Peever was a graduate of the University of Western Ontario, which only a short time ago honoured him by naming him alumni representative on the Board of Governors. He did postgraduate work at Johns Hopkins University Hospital, and worked for several years with the late Dr. W. J. Stevenson.

DR. W. E. THOMPSON, oldest medical graduate of McGill University, class of 1882, died in Los Angeles, Calif., on April 4. McGill Graduates' Society, made him an emeritus member of the society in 1947 in recognition of his long interest in the university and the society. After his graduation from McGill, Dr. Thompson was surgeon for the Canadian Pacific Railway while it was building its transcontinental route. He joined the Klondike gold rush in 1898 and was surgeon for the R.C.M.P. at Dawson City for many years. He had lived in Los Angeles for the past 20 years and was active until about a year ago. He is survived by a son and a daughter.

DR. GEORGE JOHN WEBB, of Vancouver, B.C., died recently at the age of 69. Born in Dulwich, England, he came to Canada when he was 20. Dr. Webb was an inventor at heart and had a hand in the development of diesel engines, and more recently a gas turbine. He is survived by his widow and a son.

DR. W. O. YORK, aged 79, of Edmonton, Alta., died suddenly of a heart attack in an Edmonton hospital recently. Dr. York was a native of Belfont, Arkansas. In 1906 he came to Alberta and settled in the town of Provost. Under pioneering conditions, he carried on his medical practice. He is survived by his widow, one son and three daughters.

ABSTRACTS *from current literature*

MEDICINE

The Use of Digitalis in Infants and Children.

NADAS, A. S., RUDOLPH, A. M. AND REINHOLD, J. D. L.: NEW ENGLAND J. MED., 248: 98, 1953.

Because of the paucity of information in the medical literature regarding the use of digitalis in infants and children the authors carefully studied its effect in the treatment of 41 unselected cases with congestive failure admitted to the Medical Service of the Children's Medical Centre, Boston, over the two-year period 1950-51. Congenital defects formed the etiological factor in the greater number of these patients, followed in order of frequency by myocardial disease, paroxysmal auricular tachycardia and rheumatic carditis. The small number of cases of rheumatic carditis in this group was explained by the fact that such patients were routinely sent to another institution.

Congestive heart failure occurring within the first six months of life most commonly results from congenital cardiac abnormality. Paroxysmal auricular tachycardia is the second most likely cause. Heart failure is most likely to occur after the age of two years.

The authors found that digitalis was most efficacious in congestive failure due to primary myocardial disease, paroxysmal tachycardia and rheumatic carditis. It was least effective where failure occurred on the basis of congenital cardiac abnormality, especially if cyanosis was present. Patients who exhibited electrocardiographic evidence of digitalis toxicity most readily were the ones who responded best to the drug. Dosage requirements were found to roughly approximate age and weight, children under two years of age requiring 0.02 to 0.03 mgm. digitoxin per pound for digitilization and 0.01 to 0.02 mgm. per pound body weight after the age of two.

NORMAN S. SKINNER

Some Fundamentals of Ear, Nose, and Throat Hygiene.

BARTON, R. T.: POST.-GRAD. MED., 12: 451, 1952.

The old aphorism: "Nothing in your ear smaller than your elbow," is still good advice. Where the forefinger is used to "dig out the wax in the ear" it usually packs it deeper into the canal. Cerumen may usually be removed by gentle washing with alcohol. The itchy ear should not be scratched with any available object (hairpins or match sticks) but irrigated or bathed with 70% ethyl alcohol or by oral antihistaminics. Scratching may cause painful inflammation of the ear canal. Ointments tend to accumulate in the canal, analgesics are more effective orally than locally. "Ear fungus" will be cured by 70% ethyl alcohol drops and the patient should avoid the use of soap and water in the ear. In cases of otitis externa swimming should be avoided if possible, and a person with a perforated tympanic membrane should not swim. Ear plugs are inadequate protection in swimming and hair shampooing. The most common foreign body is a cotton plug lost by the self-treated patient.

The opening aphorism is equally applicable to the nose. "Vestibularis digitorum" means inflammation of the nasal vestibule caused by one who picks his nose. This results in trauma to the nasal mucosa, with slight bleeding and scabbing. The scab is picked off and epistaxis may result. If the vibrissæ need trimming it should only be done after the scissors have been soaked in ethyl alcohol and after preparing the area by painting with aqueous zephiran or ethyl alcohol. Vasoconstriction and antihistaminics will decongest the nose as effectively when taken by mouth as when dropped into the nose, and with much less reactive vasodilatation. Postnasal drip is normal, since the cilia sweep backward toward the nasopharynx.

Antibiotic lozenges may lead to sensitivity and their result is not always successful. Gingivitis may be caused by over-zealous brushing of the teeth. Infectious mononucleosis is prone to occur in institutions and must be borne in mind with any membrane on the tonsils or throat, or in any patient with a lymphadenopathy.

J. A. STEWART DORRANCE

Incompatible Blood Transfusions.

DISCOMBE, G.: LANCET, 1: 734, 1952.

This is a report of 4,000 blood transfusions involving 10,000 bottles (5,000 litres) of blood over a 5 year period. Blood transfusion, although a life-saving measure at times, is not without risks; hepatitis occurs in 0.8%, also there is incompatibility, infection, and reaction. Whole blood may be provided in a few minutes, but there is still the risk of incompatibility, as it would take

Canad. M. A. J.
June 1953, vol. 68

3 hours to perform all the tests required to exclude all types of incompatibility. Gross blunders and incompetent personnel are the common causes of error. Administration is unsatisfactory if responsibility for the entire process of blood collecting, grouping, and issue to the wards is not centred in one person. Too many people should not be allowed to handle blood for transfusions. Technical errors may be avoided by appointing a specialist blood-grouping technician to work in close association with director. Blood should not be warmed beyond body temperature before transfusion, if so it becomes lethal. Blood which has been frozen and then thawed is unsuitable for transfusions. Small-pool plasma and dextran have obviated the need for emergency transfusions.

J. A. STEWART DORRANCE

Auricular Fibrillation.

LUNDY, C. J. AND MARCH, J. F.: POST.-GRAD. MED., 11: 449, 1952.

Auricular fibrillation is caused by multiple stimuli arising in the auricles and these stimuli spread along the muscle fibres to cause a "pot-boiling" or "delirium" of auricular activity. The circulation is inefficient in the auricles and stasis occurs in the auricular appendages and thrombi form. Auricular fibrillation may occur as a transitory arrhythmia or it may be due to extracardiac factors or heart disease. Toxic infections, coffee, nicotine, or alcohol with or without fatigue may precipitate auricular fibrillation. Excessive fatigue from overwork with or without rest may be the only demonstrable etiology. Thyroid or digitalis in excess, as well as hypoxia during anæsthesia may produce it, with or without associated heart disease. Auricular fibrillation may occur in hyperthyroidism, far advanced rheumatic heart disease, or arteriosclerotic heart disease. It is frequent in almost any form of heart disease. Treatment depends upon the treatment of the underlying or associated condition, and digitalis—rapid digitalization and maintenance. If there is no evidence of cardiac decompensation quinidene may be used in preference to digitalis; these drugs may be used in combination without the risk of pulmonary infarction. Treatment should be maintained for at least 3 weeks or longer if necessary. J. A. STEWART DORRANCE

The Use and Abuse of Ointments in Eye Disease.

MEYER, S. J. AND SCHALL, S.: POST.-GRAD. MED., 12: 552, 1952.

When it is desirable for an ointment to remain in the conjunctival sac for prolonged effect the drug should be given in a base or vehicle for which it has less affinity than for the cornea and conjunctival epithelium. The easiest method of administration of an eye ointment is to retract the lower lid and instill the ointment from a collapsible tube into the lower fornix. The physician should take particular care in instructing the patient in this procedure.

Blepharitis is treated with 10% sodium sulamyd ointment or an appropriate antibiotic three times daily. Styes—hordeola or chalazions—may be treated locally with a suitable antibiotic ointment—sulamyd or terramycin. Contact dermatitis of the lids is best treated by avoiding the sensitizing agent. Topical application of pyribenzamine ointment or 10% naftalin will relieve the itching. Conjunctivitis responds to the ointments of terramycin, aureomycin, penicillin, and streptomycin. Chemical burns of the eye should be treated initially by very thorough irrigation of the conjunctival sac and instillation of ointment. In cases of minor trauma or foreign body in the conjunctiva, an antibiotic or sodium sulamyd ointment may be instilled as prophylaxis against infection.

Keratitis is best treated by the specific antibacterial or antiviral drugs as an ointment instilled into the conjunctiva and parenterally as well. Corneal ulcer or abrasion may be treated with an antibiotic ointment and patch over the affected eye. The pain associated with this condition is best controlled by aspirin plus codeine by mouth. Inflammation of the iris and ciliary body takes a more favourable course if these structures are immobilized by the use of 1% atropine sulphate ointment or solution. Glaucoma responds to 0.25% eserine salicylate or 2% pilocarpine nitrate ointments.

J. A. STEWART DORRANCE

SURGERY

Free Autogenous Vein Graft to the Internal and Common Carotid Arteries in the Treatment of Tumours of the Neck.

CONLEY, J. J.: ANN. SURG., 137: 205, 1953.

When it is necessary to excise the carotid bifurcation in the treatment of carotid body tumours, A-V fistulas, hæmangiomas, cancer and ulceration of the carotid bulb, an end-to-end anastomosis of a free autogenous vein graft has been successful. Ligation of the internal carotid artery so often leads to hemiplegia or fatality that a serious lesion is not removed rather than take the risk, but the author describes six successful cases. Other cases are described where post-irradiation necrosis and infection combined led to failure. Thrombosis is prevented by filling the graft with a solution of heparin. In some cases hemiplegia already present was cured by vein graft. The great saphenous or superficial femoral vein were used. The procedure is described as life-saving, but further investigation is necessary to determine which patients require such a formidable operation.

BURNS PLEWES

Metabolic Rate and Thyroid Function Following Acute Thermal Trauma in Man.

COPE, O., NARDI, G. L., TUIJANO, M., ROVIT, R. L., STANBURY, J. B. AND WIGHT, A.: ANN. SURG., 137: 165, 1953.

A study of eleven severely burned patients showed that the elevated metabolic rate encountered (+30 to +60) accounts for the wasting. The thyroid gland plays no part in elevating the metabolic rate. Increased adrenal cortex activity following thermal trauma is shown to be coincidental.

BURNS PLEWES

The Ebb and Flow of the Eosinophils in the Burned Patient and Their Use in the Clinical Management.

WIGHT, A., RAKER, J. W., MERRINGTON, W. R. AND COPE, O.: ANN. SURG., 137: 175, 1953.

A prompt drop in eosinophil count occurs following burn trauma of moderate or extensive burns, but not in minor burns. The eosinophil count rises by the 3rd day if progress is satisfactory, but it is delayed by infection or operation. A failure of reappearance of eosinophils by the 3rd day is a grave prognostic sign. A secondary fall in œsinophils without burn dressings or surgical procedures heralds the development of complication such as sepsis or renal failure and should warn against additional surgery. A flood tide of eosinophils is to be expected in the later weeks or months if the patient is doing well and its absence suggests a poor prognosis.

BURNS PLEWES

The Hæmodynamics of the Surgical Patient Under General Anæsthesia.

SHACKMAN, R., GRABER, G. I. AND MELROSE, D. G.: BRIT. J. SURG., 40: 13, 1952.

A study of 32 adult patients undergoing major abdominal surgery included cardiac output by cardiac catheterization, skin and muscle blood-flows by plethysmography. Vascular resistance in skin and muscle were computed. The major circulatory adjustments which accompany

anæsthesia results in nearly half the cardiac output going to skin and muscle where profound vasodilatation occurs. It is likely that compensatory vasoconstriction occurs in the splanchnic bed.

If the operation was prolonged, skin and muscle blood-flows decreased and cardiac output fell in most patients. Prolonged splanchnic vasoconstriction and especially decreased liver blood-flow may have profound effects such as irreversible shock and varying degrees of liver dysfunction.

BURNS PLEWES

PÆDIATRICS

The Incidence and Significance of Breast Feeding in Infants Admitted to Hospital.

ASHER, P.: ARCH. DIS. CHILDHOOD, 27: 270, 1952.

A group of 1,044 infants, 16 weeks or younger admitted to Birmingham Children's Hospital were divided into "clean" or "infected", according to the disease for which the child was admitted. Clean cases were divided into "pyloric stenosis" and "other clean", and "infected" into upper and lower respiratory tract infections, diarrhœa and vomiting, and other infections. More clean cases than infected ones were breast-fed on admission, especially among those aged 5 to 8 and 9 to 12 weeks, where the difference is statistically significant. When the different groups of infection are considered separately it is seen that scarcely any babies with diarrhœa and vomiting or with upper respiratory tract infections were breast-fed. Babies with lower respiratory and other infections were more often breast-fed, though less frequently than clean cases. The child's place in the family was also studied. More children with infections had older brothers and sisters, but the difference was not significant, nor did the incidence of breast-feeding in the various disease groups vary with the place in the family. The admission of a breast-fed infant means an upheaval in the home, in some cases involving the admission of the mother as well, hence the mother would be more willing to enter hospital with her child for the treatment of pyloric stenosis than otitis media. At ages 5 to 8 weeks the percentage of cases of pyloric stenosis breast-fed on admission was nearly six times that of infected cases. A considerable number of babies with pyloric stenosis had been recently weaned with the mistaken idea that the mother's milk was the cause of the vomiting; were it not for this still more of these would have been breast-fed. It is instructive that upper respiratory tract infection, like infantile diarrhœa, should be so largely confined to the formula-fed. As the formula-fed baby may be fed by numbers of different people it runs more risk of contracting an intercurrent infection, but impaired nutrition or delayed immunity may also play a part. This may be only surmise; the important point in the figures obtained is the considerably increased dangers of at least certain infections to which the artificially-fed baby is exposed.

J. A. STEWART DORRANCE

Effects of Citrate Feeding on Rachitic Infants.

HARRISON, H. E. AND HARRISON, H. C.: J. PEDIAT., 41: 756, 1952.

The authors report on 3 rachitic infants treated with citrate mixture without vitamin D. The studies included determinations of serum calcium, serum phosphorus, serum citrate, and serum phosphatase, and the daily determinations of the excretion of calcium, phosphorus, and citrate in the urine. The long bones were examined by x-ray to study the deposition of bone salt in the rachitic cartilage and this was correlated with the concentrations of calcium and phosphorus in the serum. The citrate solution given to the infants was made up of an equimolar mixture of citric acid and trisodium citrate; this was added to the milk feedings in amounts ranging from 30 to 60 mm. of citrate per day.

In two infants with advanced florid rickets there was a marked drop of serum calcium to hypocalcæmic levels, yet there was x-ray evidence of deposition of bone salt in the rachitic cartilage. The urinary excretion of calcium remained low during this period of citrate treatment while the excretion of phosphorus was unchanged. The concentrations of serum citrate were not consistently higher during citrate treatment, but rose after vitamin D was given. A third infant with early rickets and tetany showed a decrease in serum calcium when citrate was given; the concentration of phosphate and of citrate rose to normal values when the alkaline phosphatase activity in the serum decreased. There was normal growth and calcification of the bones during the period of citrate therapy without vitamin D. The antirachitic effect of citrate feeding is due to a local action on the proliferating cartilage and bone matrix causing an increased calcifiability; also there is the possibility of an interrelationship between citrate metabolism and the physiological effects of vitamin D.

J. A. STEWART DORRANCE

Endotracheal Insufflation with Oxygen in the Treatment of Asphyxia Neonatorum.

O'BRIEN, D. AND ROBERTS, H.: BRIT. M. J., 2: 963, 1952.

The authors report on 66 infants with asphyxia neonatorum from 5,458 deliveries. Where respiration had not started by 3 minutes after birth, a small metal airway was inserted and the infant rocked. The group reported consists of those who after 3 minutes of rocking (6 minutes after birth) still showed no response. The pharynx was then aspirated under direct vision and an endotracheal tube inserted. This was attached to a Y-shaped metal connection to enable the operator to apply oxygen at pressures of 20 mm. of water by occluding the open end for periods of 3 seconds between 10 and 15 times per minute. The optimum rate of flow was 1.5 litres per minute, this was found to facilitate the insertion of the tube through the glottis. Of the 66 infants thus intubated 14 died, the remaining 52 made uninterrupted recoveries. The authors feel that this method helps to expand atelectactic lungs as long as the oxygen is administered, but the condition reverts to collapse when the procedure is discontinued. This procedure, though momentarily expanding the lung and alveoli, does not appear to permanently modify the physical state of the unexpanded newborn lung.

J. A. STEWART DORRANCE

THERAPEUTICS

N-Allyl Normorphine: an Antagonist to the Opiates.

ECKENHOFF, J. E., HOFFMAN, G. L. AND DRIPPS, R. D.: ANÆSTHESIOLOGY, 13: 242, 1952.

On the basis of a report that N-allyl normorphine reversed respiratory depression produced in animals by large doses of morphine, the authors became interested in this drug and administered it to about 400 patients.

In patients who had received 20 to 90 mgm. of morphine sulphate or 200 to 600 mgm. of meperidine, the intravenous administration of 5 or 10 mgm. of n-allyl normorphine doubled or tripled the respiratory rate and increased respiratory minute volume as much as 250%. These effects reached a peak within one to two minutes, then declined gradually, but both values remained above the depressed levels for the sixty minute period of observation. In addition to respiratory stimulation, normorphine caused an elevation of blood pressure when it had been depressed by the narcosis. It was noted that, in the doses used, the antagonist seemed to have little awakening effect. An unexpected action was that when given in 5 or 10 mgm. doses to normal volunteer subjects, the

Canad. M. A. J.
June 1953, vol. 68

drug produced a depression of respiration and blood pressure. It was found that n-allyl normorphine was also an effective antagonist in depression produced by pantopon, dilaudid and methadone. While the drug is a specific antagonist to depression produced by the opiates, it is not effective against depression produced by other depressants of the central nervous system. In the doses which the author employed, it was found to be useless in depression by cyclopropane, ethyl ether, thiopental and secobarbital sodium. The drug was used clinically four times in the treatment of actual opiate overdosage with a satisfactory result in every case.

In 255 obstetrical patients, n-allyl normorphine shortened significantly the interval between the delivery of the chin and the infant's first gasp or establishment of respiration when the child was born of a mother in a moderate or deep state of depression caused by analgesics and sedatives. The drug is thus valuable in the prevention and the treatment of neonatal asphyxia and depression, bearing in mind that it is not an effective antagonist to barbiturates. Also, depression basically due to trauma of delivery, nuchal cord with resultant asphyxia, pre-mature placental separation, intra-uterine pulmonary infection, or other similar conditions obviously cannot be affected by the drug. B. L. FRANK

Heparin in the Treatment of Advanced Peripheral Atherosclerosis.

ENGELBERG, H. AND MASSELL, T. B.: AM. J. M. SC., 225: 14, 1953.

A series of thirteen patients with severe peripheral atherosclerotic disease received intermittent treatment with heparin for six months. At the end of this period the authors felt that the definite objective and subjective improvement noted in most of the cases justified a preliminary report of their observations. Heparin was given in doses of 100 mgm. intravenously two or three times weekly, using a 25 or 26 gauge needle. In several cases heparin was administered daily for the first month. In all instances, the anticoagulant effect persisted for four to seven hours after the injection. Even after six months of heparin therapy clotting times returned to normal within four to seven hours after each dose of heparin. There were no serious toxic reactions, nor any hæmorrhage.

As the two most striking manifestations of arterial insufficiency in the lower extremities are intermittent claudication at first, and gangrene finally, it was decided to test the efficacy of therapy by measuring the blood flow through muscle and digits. Compared with other forms of treatment, such as sympathectomy, the changes were striking. Generally, the improvement in the peripheral circulation occurred gradually and progressively over the period of observation. In eight out of ten cases there was marked improvement in walking tolerance varying from 17 to 800%. Clinically, 11 of the 33 patients had intermittent claudication and nine of these improved symptomatically. This occurred usually after one to several months of therapy, and, in general, paralleled the increase in muscle flow. When therapy was stopped in four patients the symptoms recurred in four to eight weeks. B. L. FRANK

Banthine Therapy for Enuresis.

O'MALLEY, J. F. AND OWENS, R. H.: MISSOURI MED., 50: 188, 1953.

Enuresis has been described as repeated involuntary micturation after the age of three. True enuresis is distinguished from pseudo-enuresis by the fact that in the latter, the child has developed normal control but reverts to bed wetting at various intervals. Since it is thought that the patient suffering from enuresis has an irritable bladder with a tendency toward uninhibited contractions, the authors chose Banthine, which has been used in the treatment of irritable bladders, cystitis and Hunner's ulcer. Banthine, by inhibiting the local reflex, allows the bladder to increase its capacity and to accommodate a larger volume of urine without an increase in intravesical pressure; in this way, adequate inhibition is said to be maintained at the reflex level in spite of poor cerebral inhibition.

Eleven cases of long standing enuresis were treated with 25 to 75 mgm. of banthine at bed time. The ages of the patients ranged from five to fifteen. In most cases, bed wetting was eliminated after a short course of therapy. Three cases relapsed, but by increasing the dosage of banthine, these were also improved. The authors feel that with Banthine the habit of continence can be established by allowing the bladder to accommodate larger amounts of fluid without reflex voiding. The self-assurance gained by the patient appears to improve the psychologic aspect of the problem.

B. L. FRANK

OBSTETRICS AND GYNÆCOLOGY

Pregnancy Complicated by Diabetes Mellitus.

NELSON, H. B., GILLESPIE, L. AND WHITE, P.: OBST. AND GYNEC., 1: 219, 1953.

One hundred and sixty-three diabetic pregnancies were observed over a two-year period. A pregnancy risk classification of diabetes is presented.

Fourteen therapeutic interruptions were performed in patients with severe retinal or renal disease. Twenty-one patients either aborted spontaneously or delivered premature nonviable infants. There were 128 viable pregnancies. Among the viable pregnancies, the live-born rate was 98.4%, and the fetal survival rate 90%.

ROSS MITCHELL

An Analysis of Five Hundred and Forty-Seven Midforceps Operations.

DECKER, W. H., DICKSON, W. A. AND HEATON, C. E.: AM. J. OBST. AND GYNEC., 65: 294, 1953.

An analysis of 547 midforceps deliveries is presented with a gross fetal mortality of 6.58% and a corrected fetal mortality of 4.75%. Factors responsible for the lowering of the fetal mortality rates in infants delivered by midforceps are presented. The importance of selecting the proper forceps for each delivery is stressed. Emphasis is placed on the evaluation of the bony pelvic architecture in determining the means by which delivery will be accomplished.

Midforceps can be a safe method of delivery when all factors are considered and the more difficult and hazardous cases are eliminated. ROSS MITCHELL

Circumvallate Placenta.

PAALMAN, R. J. AND VANDER VEER, C. G.: AM. J. OBST. AND GYNEC., 65: 491, 1953.

Experience with 41 cases of circumvallate placenta in 8,562 consecutive deliveries has been presented to demonstrate the clinical significance of this condition in obstetrics. Intermittent vaginal bleeding, hydrorrhœa, and uterine contractions are the common symptoms which lead one to suspect the presence of circumvallate placenta. The predelivery diagnosis is most often confused with placenta prævia and abruptio placentæ. Management of patients with circumvallate placenta is primarily expectant, although bleeding in some cases demands definite treatment. The vital threats to the mother are hæmorrhage, infection and third-stage complications. The fetal survival rate is low, due principally to late abortions and premature labours. ROSS MITCHELL

Bacteriology of Vaginal Flora After the Use of Internal Tampons.

BRAND, E. S.: BRIT. M. J., 1: 24, 1952.

A group of 100 women used vaginal tampon for menstrual protection in 1 to 10 successive periods. Smear and culture showed no appreciable changes in the bacterial flora of the vagina, when taken before and after each period. Healing of cervical erosion was not adversely affected. There was no evidence of local irritation or inflammation, nor was there alteration of the pH or the glycogen content of the epithelial cells. A retained tampon may cause damage to the cervix or vagina and soiled tampons should be removed before the insertion of a fresh one. Removal is particularly important at the end of a menstrual period. J. A. STEWART DORRANCE

Relationship of Specific Attitudes and Emotions to Certain Bodily Diseases.

GRACE, W. J. AND GRAHAM, D. T.: PSYCHOSOMATIC MED., 14: 143, 1952.

One hundred and twenty-eight patients who had one or more of twelve symptoms or diseases were studied in the out-patient department of the New York Hospital-Cornell Medical Centre. The authors found that there was associated with each condition a definite, completely conscious attitude toward the precipitating situation. This attitude was peculiar to the condition, which did not occur without it. "Attitude" is defined as what the patient felt was happening to him and what he wanted to do about it at the time of the occurrence of the symptom.

The results are interesting. All patients with the same symptom-complex described their attitudes toward the situation which precipitated it in essentially the same way. The following attitudes and physiological disturbances were found to be associated: (1) Urticaria occurred when an individual saw himself as being mistreated. (2) Eczema occurred when an individual felt he was being interfered with or prevented from doing something and could think of no way to deal with the frustration. (3) Cold and moist hands occurred when an individual felt that he should undertake some kind of activity, even though he might not know precisely what to do. (4) Vasomotor rhinitis occurred when an individual was facing a situation with the wish that he didn't have to do anything about it. (5) Asthma occurred in association with attitudes exactly like those in (4), but presumably feelings were more intense. (6) Diarrhœa occurred when an individual wanted to be done with a situation or to have it over with, or to get rid of something or somebody. (7) Constipation occurred when an individual was grimly determined to carry on even though faced with a problem he could not solve. (8) Nausea and vomiting occurred when an individual was thinking of something which he wished had never happened. (9) Duodenal ulcer occurred when an individual was seeking revenge. (10) Migraine headache occurred when an individual had been making an intense effort to carry out a definite planned program, or to achieve some definite objective. The headache occurred when the effort had ceased. (11) Arterial hypertension occurred when an individual felt that he must be constantly prepared to meet all possible threats. (12) Low back pain occurred when an individual wanted to carry out some action involving movement of the entire body.

The authors comment, following Cannon and Wolff, that in many cases the attitude can be considered as a description of the function of the physiological process with which it is associated. In any situation which precipitates illness the patient has an attitude or way in which he perceives his own position in the situation and the action, if any, which he wishes to take to deal with it. The accompanying bodily changes may give rise to "symptoms", and if they recur or persist, are said to represent a "disease". Most of the common diseases can be viewed as the outcome of physiological adjustments.

F. W. HANLEY

ANÆSTHESIA

The Use of Parenteral Dramamine to Control Postoperative Vomiting: A Report of 1,192 Cases.

MOORE, D. C., ANDERSON, LEG., WHEELER, G. AND SCHEIDT, J.: ANÆSTHESIOLOGY, 13: 354, 1952.

The over-all percentage of postoperative vomiting was calculated for the four month period prior to the use of dramamine as a control. Since the authors found the results with dramamine so satisfactory during the six months following the control series, they stopped the parenteral administration of dramamine again for a two month period in order to obtain another control series.

All patients received 50 mgm. of dramamine intramuscularly on call to surgery, 50 mgm. of dramamine intramuscularly on return from surgery, and then 50 mgm. intramuscularly every four hours for four doses. The authors felt that nausea was a "psychic" response, the degree of which was difficult to evaluate; they, therefore, restricted their study to evaluation of vomiting. Vomiting was defined as the emesis of any material which measured 50 c.c. or more in volume. Spitting up of mucus or bile was not considered as vomiting.

In the first control series of cases the over-all incidence of vomiting was 21.27%. In the second control series, which was conducted approximately six months later, the incidence of vomiting was 25.3%, giving an average for both series of 22.2%. When dramamine was given as outlined above, the incidence of vomiting fell to 11.2%. Analyzing this over-all incidence of vomiting, it was found that in caudal, sodium pentothal and cervical block procedures there was a more marked reduction. In the anæsthetic procedures used for most major operations of the abdomen, head, neck, thorax and genito-urinary system the incidence of vomiting remained between 10 and 20%. Postoperative vomiting caused by motion appeared to be controlled by dramamine but variable conditions such as personnel involved, the site of operation and other factors seemed to play a definite part. The intramuscular injection of dramamine did not cause any tissue reaction in the cases studied.

B. L. FRANK

INDUSTRIAL MEDICINE

Labour Turnover and Absence in Great Britain Under Full Employment.

BROWN, E. H. P.: NATURE, 168: 993, 1951.

The rates of labour turnover and absence in Great Britain have been higher under full employment than before the Second World War and that they constitute a substantial obstacle to industrial efficiency, is a common belief. Until recently, however, there have been no records in sufficient detail to reveal the course and causes of turnover and absence in a particular setting. For that reason the two monographs reviewed in this article are important. "Labour Turnover under Full Employment" by Dr. Joyce R. Long and "Absence under Full Employment" by Hilde Behrend, were published recently by the University of Birmingham Faculty of Commerce and Social Science. They report and analyze the experience of factories in the Midlands, employing between them upwards of 60,000 persons mostly in engineering and metal working.

That the rate of turnover depends on the type of worker rather than on the type of firm and that some types of worker have high rate of turnover whereas the majority are stable, is the main finding of Dr. Long's study. The report indicates the characteristics associated with types whose turnover-rates are habitually high as chiefly youth, lack of skill and coming to work from a distance. It shows also that women usually show higher rates than men; much of this however, seems to depend on lack of skill rather than on sex. Conditions of employment such as size of the firm, the level of pay and the working environment showed no clear association with the turnover-rate.

Canad. M. A. J.
June 1953, vol. 68

A means for measuring controllable and uncontrollable absence separately is proposed in Miss Behrend's study of absence. Her investigation revealed that a given plant or department seems to have a characteristic and persistent weekly pattern. She suggests that it should take its Friday-rate as the measure of the uncontrollable absence specific to it, and the excess of absence on other days as the measure of controllable absence. She discusses also the significance of a "Blue Monday" index.

Each monograph includes what evidence there is for the difference between recent working and that of the interwar years. The evidence is scanty but it shows less difference than common report suggests.

MARGARET H. WILTON

Phosphate Ester Poisoning, a New Problem for the Internist.

HAMBLIN, D. O. AND MARCHAND, J. F.: ANN. INT. MED., 36: 50, 1952.

Because of their exceptional effectiveness as agricultural insecticides, several esters of phosphoric acid have been manufactured and applied on an expanding scale. In this article the authors stress the need for intelligent care in their handling and use. They maintain that in skilled hands, these insecticides have done an important job and should continue to do so safely until the day arrives when a truly less toxic but equally effective insecticide can be found. Special reference is made to parathion. A number of parathion poisonings, several of them fatal, have already occurred in this country. The safety record, however, has improved, with expanding knowledge of the biologic properties of the material and of the necessary protective measures.

The authors outline briefly the history of the discovery of the cholinesterase-inhibiting action of certain phosphate esters and, directions for their use. Where sprays or dusts have been applied according to schedules approved by the U.S. Department of Agriculture, there has been no problem with respect to residue toxicity. Mention is made also of the so-called nerve gases. Details are given also regarding the pharmacology of the group as a whole. With regard to mode of action, no clearcut indications of any action other than that on the cholinesterase enzyme system have yet been available from published reports of animal experiments or human experience.

The signs and symptoms of phosphate ester poisoning, as observed in man, have included giddiness, headache, confusion, weakness, nausea, vomiting, cramps, diarrhœa, sweating, pupillary constriction, occasionally an irregular pulse, and often a sense of constriction in the chest. Absorption through the skin is rapid although no topical actions on the skin except local warmth and sweating, have occurred. Symptoms develop only during, or within a few hours of, exposure and subside completely within a day or two. There is, however, a danger from repeated small exposures and from new exposures during convalescence from a previous poisoning.

In order to prevent accidental poisoning, meticulous attention to protective measures against skin contact or inhalation of airborne sprays, is required. Should there be even a remote risk of exposure as in manufacturing or spray operations, a regular testing schedule for plasma and red cell samples is also essential.

When acute poisoning does occur, the problem of effective therapy is urgent and immediate as the outcome may be determined in a matter of hours or even minutes. The specific antidote is atropine, given at once, by any convenient route and in large amounts. Dosage and general supportive measures are given. The authors stress the importance of a readily accessible supply of atropine with proper directions for emergency use, for the safety of persons handling these materials. They caution, however, against the prophylactic use or other misuse of such supplies. Any man taking atropine prior to risking exposure may thereby deprive himself of the warning symptoms which might otherwise permit the timely application of therapeutic measures.

MARGARET H. WILTON

FORTHCOMING MEETINGS

CANADA

CANADIAN SOCIETY OF MICROBIOLOGISTS, Annual Meeting, Ontario Agricultural College, Guelph, Ont. (Dr. N. E. Gibbons, Division of Applied Biology, National Research Council, Ottawa 2, Ont.) June 4-6, 1953.

CANADIAN ASSOCIATION OF PHYSICAL MEDICINE, Annual Meeting, Osler Hall of the Academy of Medicine, Toronto, Ont. June 5, 1953.

THE SOCIETY OF OBSTETRICIANS AND GYNÆCOLOGISTS OF CANADA, Annual Meeting, The Thousand Island Club, Alexandria Bay, N.Y. (Dr. G. A. Simpson, Secretary, Maternity Pavilion, Royal Victoria Hospital, Montreal) June 5-7, 1953.

CANADIAN UROLOGICAL ASSOCIATION, Annual Meeting, Minaki Lodge, Ont. (Dr. S. A. MacDonald, Secretary, 1414 Drummond St., Montreal) June 10-12, 1953.

CANADIAN MEDICAL ASSOCIATION, Annual Meeting, Royal Alexandra Hotel, Winnipeg, Man. (Dr. T. C. Routley, 135 St. Clair Ave. W., Toronto 5, Ont.) June 15-19, 1953.

CANADIAN ASSOCIATION OF RADIOLOGISTS, Winnipeg, Man. (Dr. Jean Bouchard, Secretary, 1555 Summerhill Ave., Montreal 25, Que.) June 15-16, 1953.

CANADIAN ASSOCIATION OF PATHOLOGISTS, Winnipeg, Man. (Dr. D. W. Penner, Secretary, Winnipeg General Hospital, Winnipeg, Man.) June 15-16, 1953.

CANADIAN RHEUMATISM ASSOCIATION, Winnipeg, Man. (Dr. T. E. Hunt, Secretary, P.O. Box 277, Regina, Sask.) June 15-16, 1953.

CANADIAN ACADEMY OF ALLERGY, Winnipeg, Man. (Dr. T. H. Aaron, Secretary, 502 McLeod Bldg., Edmonton, Alta.) June 16, 1953.

CANADIAN PSYCHIATRIC ASSOCIATION, Winnipeg, Man. (Dr. J. P. S. Cathcart, Secretary, 183 Metcalfe St., Ottawa, Ont.) June 16, 1953.

CANADIAN HEART ASSOCIATION, Winnipeg, Man. (Dr. John Keith, Secretary, Hospital for Sick Children, 555 University Ave., Toronto, Ont.) June 16, 1953.

CANADIAN MEDICAL PROTECTIVE ASSOCIATION, Winnipeg, Man. (Dr. T. L. Fisher, Secretary, 180 Metcalfe St., Ottawa, Ont.) June 18, 1953.

THE FEDERATION OF MEDICAL WOMEN OF CANADA, Winnipeg, Man. (Dr. J. G. Bellamy, Secretary, 2455-29th Ave. S. W., Calgary, Alta.) in June but dates still uncertain.

CANADIAN NEUROLOGICAL SOCIETY, Winnipeg, Man. (Dr. Allan Walters, Secretary, Medical Arts Bldg., Toronto, Ont.) June 20-21, 1953.

INTERNATIONAL CONVENTION OF X-RAY TECHNICIANS, Royal York Hotel, Toronto, Ont. (Miss Beatrice Hurley, R.T., Registrar, St. Catherine Hospital, East Chicago, Indiana) June 28 - July 2, 1953.

INTERNATIONAL PHYSIOLOGICAL CONGRESS, 19th Congress, Montreal, Canada (Miss MacCallum, Donner Bldg., McGill University, Montreal), September 1-5, 1953.

BRITISH COLUMBIA DIVISION, C.M.A., Annual Meeting, Vancouver, B.C. (Dr. G. Gordon Ferguson, Exec. Secretary, 1807 West 10th Ave., Vancouver, B.C.) September 21-25, 1953.

CONGRESS OF ANÆSTHETISTS, 28th Annual Congress, Chateau Frontenac, Quebec, P.Q., (Laurette McMechan, Assistant Executive Secretary, 318 Hotel Westlake, Rocky River 16, Ohio) October 26-29, 1953.

UNITED STATES

AMERICAN MEDICAL ASSOCIATION, Annual Session, New York, N.Y. (Dr. George F. Lull, 535 N. Dearborn St., Chicago 10, Ill.), June 1-5, 1953.

AMERICAN COLLEGE OF CARDIOLOGY, 2nd Annual Convention, Hotel Statler, Washington, D.C. (Dr. Philip Reichert, Secretary, 480 Park Ave., New York 22, N.Y.) June 7-9, 1953.

INTERNATIONAL CONGRESS OF ELECTROENCEPHALOGRAPHY AND CLINICAL NEUROPHYSIOLOGY, Boston, Mass. (Dr. R. S. Schwab, Secretary-General, Mass. General Hospital, Boston 14, Mass.) August 18-21, 1953.

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY, 17th Congress, Waldorf-Astoria Hotel, New York City, N.Y. (Dr. W. L. Benedict, Secretary General, 100-1st Ave. Bldg., Rochester, Minn.) September 12-17, 1953.

AMERICAN MEDICAL WRITERS' ASSOCIATION, 10th Annual Meeting, Springfield, Ill. (Dr. Harold Swanberg, Secretary, 209-224 W. C. U. Bldg., Quincy, Ill.) September 23, 1953.

NATIONAL GASTROENTEROLOGICAL ASSOCIATION, 18th Annual Convention, Los Angeles, Calif. (Dr. Samuel Weiss, 146 Central Park West, New York 23, N.Y.) October 12-14, 1953.

OTHER COUNTRIES

INTERNATIONAL CONGRESS OF OTORHINOLARYNGOLOGY, 5th Congress, Amsterdam, Holland (Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam) June 8-15, 1953.

PAN AMERICAN CONGRESS OF THE MEDICAL PRESS, Buenos Aires, Argentine (Secretaria del Congress, 763 Uriburu, Buenos Aires, Argentine) July 12-16, 1953.

BRITISH MEDICAL ASSOCIATION, Cardiff, S. Wales (Dr. A. MacCrae, Secretary, B.M.A. House, Tavistock Square, London W. C. 1, England) July 13-17, 1953.

INTERNATIONAL CONGRESS OF RADIO-BIOLOGY, Copenhagen, Denmark (Prof. F. Norgaard, Secretary General, Oster Voldgade 10, Copenhagen K, Denmark) July 14-25, 1953.

INTERNATIONAL FEDERATION OF HOSPITALS, Congress, Brussels, Belgium (Capt. J. E. Stone, 10 Old Jewry, London, E.C.2, England) July 15-21, 1953.

INTERNATIONAL CONGRESS OF RADIOLOGY, 7th Congress, Copenhagen, Denmark (Prof. F. Norgaard, 10 Oster Voldgade, Copenhagen K), July 19-25, 1953.

INTERNATIONAL CONGRESS ON MEDICAL LIBRARIANSHIP, First Congress, London, England (Mr. W. R. Le Fanu, Chairman, c/o London School of Hygiene and Tropical Medicine, Keppel St., London, W.C.1), July 20-25, 1953.

INTERNATIONAL CONGRESS FOR HISTORY OF SCIENCE, Jerusalem, Israel (Prof. F. S. Bodenheimer, President, Hebrew University, Jerusalem, Israel) August 3-7, 1953.

WORLD CONFERENCE ON MEDICAL EDUCATION, British Medical Association House, Tavistock Square, W.C.1, London, England (Dr. Louis H. Bauer, The World Medical Association, 2 East 103rd St., New York 29, N.Y.) August 24-29, 1953.

INTERNATIONAL CONGRESS OF TROPICAL MEDICINE AND MALARIA, Istanbul, Turkey (Prof. Dr. Ihsan Sükrü Aksel, General Secretary, Tunel Meydam, Beyoglu, Istanbul, Turkey) August 28-September 4, 1953.

WORLD MEDICAL ASSOCIATION, 7th General Assembly, The Hague, Amsterdam, Holland (Dr. Louis H. Bauer, Secretary-General, 2 East 103rd St., New York 29, N.Y.) August 31-September 6, 1953.

INTERNATIONAL CONGRESS OF MICROBIOLOGY, 6th Congress, Rome, Italy (Dr. N. E. Gibbons, Secretary, Canadian Society of Microbiology, Division of Applied Biology, National Research Council, Ottawa 2, Ont.) September 6-12, 1953.

WORLD CONFEDERATION FOR PHYSICAL THERAPY, 1st Congress, London, England (Miss M. J. Neilson, Secretary, Chartered Society of Physiotherapy, South Tavistock Square, London, W.C.1, England) September 7-12, 1953.

INTERNATIONAL SOCIETY OF SURGERY, CONGRESS, Lisbon, Portugal. (Dr. L. Dejardin, General Secretary, rue Belliard, Brussels, Belgium) September 14-20, 1953.

INTERNATIONAL CONGRESS OF PÆDIATRICS, Havana, Cuba (Prof. Felix Hurtado, President, 5a Avenue 124, Miramar, Havana, Cuba) October 12-17, 1953.

NEWS ITEMS

ALBERTA

Dr. A. B. King of Vermilion attended the University Hospital surgical rounds before returning to his active practice in the former town.

The May issue of the *Alberta Medical Bulletin* is the Coronation number which is in keeping with the crowning of Queen Elizabeth II on June 2, 1953. Special articles have been written showing the relationship of Royalty and the medical profession through the years. The Editorial Board wish to thank those authors who contributed to this issue.

The members of the medical profession in Alberta join with the rest of Canada in wishing Queen Elizabeth II and her Consort the Duke of Edinburgh long life and happiness during Her Majesty's reign.

The Calgary hospitals have accepted a goodly number of interns this spring. There is much valuable experience and knowledge to be obtained at these hospitals which are staffed with excellent teachers and fully qualified doctors. This move has decreased the number who generally remain in Edmonton hospitals; thus the ever-changing face of medicine is evident, which is all to the good.

Dr. Guy Morton of Edmonton has returned from a sojourn to California for a month. Dr. Morton heads the Department of Neurosurgery at the University hospital.

W. C. WHITESIDE

BRITISH COLUMBIA

The Council of the College of Physicians and Surgeons held their biennial election in April, to replace those members whose four-year term was up.

The retiring members were Drs. George Davidson and D. H. Williams of Vancouver, Dr. A. C. Nash of Victoria, and Dr. F. M. Auld of Nelson. Elected in 1951 for four

Canad. M. A. J.
June 1953, vol. 68

years, and remaining on the Council are Drs. Roy Huggard and J. H. MacDermot, of Vancouver, Dr. L. Chipperfield of New Westminster, Dr. L. T. Maxwell of Prince George, and Dr. F. M. Bryant of Victoria. A new District, number 6, comprising the upper half of Vancouver Island has been created, and the member for this District is Dr. Meneely of Nanaimo.

New members (1953-7) are Drs. W. Elliott Harrison and R. G. Langston of Vancouver, Dr. R. Scott-Moncrieff of Victoria, and Dr. D. J. Crawford of Trail, who replaces Dr. Auld.

Dr. Roy Walker was elected President of Council. Vice-President is Dr. Roy Huggard, and Dr. Chipperfield is Treasurer.

The Vancouver Medical Association will hold its Annual meeting and Election of Officers on May 5 and the following have been nominated as officers: President—Dr. Don S. Monroe; Vice-President—Dr. Howard Black; Hon. Treasurer—Dr. Geo. E. Langley; Hon. Secretary—Dr. Sidney F. Hobbs; To Board of Trustees—Dr. Wallace J. Dorrance; Editor of *Bulletin*—Dr. J. H. MacDermot; Executive Members—Drs. George F. Elliott, R. A. Gilchrist and A. F. Hardyment.

St. Paul's Hospital School of Nursing held its Annual Graduation ceremonies on April 29 in the Georgia Auditorium. One hundred and two graduates received their diplomas. The meeting was addressed by Mayor Hume, and Dean G. F. Curtis of the University of British Columbia Law School gave the main address. St. Paul's has graduated more than 2,200 nurses since its nursing school was opened in 1907.

New Provincial Public Health Laboratories are to be erected in Vancouver, to replace the old buildings on Hornby Street, where for many years excellent work has been carried on under very discouraging conditions, and in very cramped and inconvenient quarters. Dr. G. F. Amyot, deputy Minister of Health made this announcement recently. The new buildings will adjoin the Tuberculosis Centre on Willow Street.

An annual award of $300.00 has been made for the foundation of a Lectureship in Medical Research through the Alumni U.B.C. Development Fund. This was made possible through the generosity of Mr. E. A. Simmons, whose name will be given to the Lectureship.

The North Okanagan District had 848 cases of communicable disease in 1952. Of these only three were polio. The report was made to the annual meeting of the Union Board of Health, which operates in the North Okanagan.

The travelling chest clinic did excellent work, taking some 700 chest x-rays in the various centres, Vernon, Armstrong, Enderby, Salmon Arm, Revelstoke, Coldstream and Spallumacheen. 850 births were recorded in the District. 667 immunizations were given, and a great deal of work has been done in connection with improvements in milk-supply, water supply, sewerage administration and so on. Inspection of plumbing, meat inspection have also come under the activities of the Union Board of Health.

J. H. MACDERMOT

MANITOBA

Dr. H. C. Stevenson was invested as an officer of the Order of the British Empire by the Queen at Buckingham Palace recently. The award of the O.B.E. was made because of Dr. Stevenson's bravery for evacuating wounded under fire when he was serving as a captain in the R.C.A.M.C. in 1951 on the Korean front. Dr. Stevenson is taking a postgraduate course at Dublin before resuming his work as municipal doctor at Minnedosa. He is a son of Dr. W. F. Stevenson of Belmont.

A coroner's jury investigating the death of a man in a steam bath has recommended closer supervision of such baths. Among their recommendations was that occupants be checked periodically, that steam valves should be within easy and safe reach and supplied with wooden handles, and that adequate ventilation be provided.

A railway car serving as a mobile Red Cross nursing station will be placed in operation on the Hudson's Bay line this summer. It will provide clinic facilities for outpatients, living quarters for a nurse, and two beds to be used in case of emergency. The car will move between Waboden and Gillam making routine trips during the summer. In winter it will be stationed alternately in the two towns. Hitherto, the area had to depend on hospitals at either end of the 500 mile strip at Churchill or The Pas. It will be operated and staffed by the Red Cross with the Provincial Department of Health and Welfare paying half the cost of administration. The nurse in charge will also serve as a public health nurse.

Winnipeg hospitals are to expand to the amount of $22,000,000. On May 15 the Joint Hospital Building fund for $8,000,000 worth of additions to the Winnipeg General and Children's hospitals will appeal to the public for $3,000,000. A new $4,500,000 wing for Deer Lodge Hospital will be erected. Misericordia is planning a new wing and a nurses' home. Steel for the eight-storey addition to St. Boniface hospital has been erected.

Col. Carl Graham Wood, command medical officer for the Prairie Command, has been appointed a serving brother for the Order of the Hospital of St. John of Jerusalem.

Dr. R. M. Cherniak, at present lecturer in the department of physiology and medical research, University of Manitoba, has received a fellowship grant of $3,900 from the Life Insurance Medical Research Fund. This will be used in the field of cardio-pulmonary research at Johns Hopkins University under the guidance of Dr. Richard L. Riley. Dr. Cherniak graduated from the University of Manitoba in 1948. For three years he worked on a fellowship at the Winnipeg General Hospital and in 1951-52 he studied under Dr. A. Barach on a fellowship at Columbia University, New York.

On April 9, the ratepayers of Selkirk, St. Andrews and St. Clements voted in favour of a money by-law of $243,000 covered by debentures to provide a new 60-bed hospital at Selkirk. Cost of the hospital is estimated at $426,000 with contributed grants of $180,000 shared by the Dominion and Provincial governments. The Manitoba Pool Elevators will contribute another $3,000. In addition to the 60 beds, the new hospital will have 18 bassinets in cubicles, two labour beds, accommodations for the Selkirk health unit, the Selkirk laboratory and x-ray units and a new home for 30 nurses.

ROSS MITCHELL

NEW BRUNSWICK

The King's County Medical Society Meeting at Sussex on March 24 heard Dr. H. Rosen discuss "The Treatment of Fracture-Dislocations of the Cervical Spine". At their April meeting, Dr. G. E. Maddison and Dr. Lachlan MacPherson read papers on "Pulmonary Tuberculosis" and "Differential Diagnosis of Allied Chest Conditions".

Dr. S. Silver was recently appointed chief of the E.E.N.T. Service at the Saint John General Hospital. Dr. Silver is certificated both in Otolaryngology and Ophthalmology and has been attached to the hospital staff

for some years. His appointment followed the resignation of Dr. R. T. Hayes as chief of the E.E.N.T. Service to accept an appointment to the Board of Commissioners of the hospital, where he has served as a clinician since his graduation.

A considerable group of staff doctors of the Saint John General Hospital have recently received certifications in their specialties. This number includes: Dr. M. I. Polowin, Surgery; Dr. S. Silver, Ophthalmology and Otolaryngology; Dr. D. R. MacRae, Otolaryngology and Ophthalmology; Dr. D. F. Sutherland, Obstetrics and Gynæcology; Dr. K. Seaman, Orthopædic Surgery; Dr. F. H. George, Internal Medicine; and Dr. J. L. Guravich, Internal Medicine.

Over the past several months, Dr. J. R. Nugent, a member of the Medical Advisory Committee of the N.B. Cancer Society and Chairman of the Clinical Advisory Committee of the N.B. Department of Health Cancer Program has addressed many lay groups on Cancer problems. His field has covered much of the southern part of New Brunswick and the numbers attending these addresses has been large.

The annual meeting of the Medical Council of New Brunswick was held on March 26 in Saint John. Dr. G. L. Dumont of Campbellton, the president, presided over a full attendance of Council. Most of the meeting was devoted to correspondence on, and a discussion of enabling certificates, particularly as they applied to foreign physicians. Progress was made in crystallization of opinions and at the next meeting it is hoped to implement some useful regulations on this subject.

Dr. H. R. Bryant of Sussex addressed a meeting of the King's County Branch of the N.B. Cancer Society at the Sussex Hospital in April. His remarks were followed by the showing of one of the recent cancer films.

Dr. R. A. H. MacKeen as a medical member of the Arthritis Association, appeared before the Saint John Trades and Labour Congress and discussed arthritis before a large and appreciative audience early in April. At a later date he was guest speaker at the Kiwanis Club in Saint John, when he spoke on Cancer. At this meeting he was introduced by Dr. Geo. White, Kiwanis Lieutenant-Governor of the district. A. S. KIRKLAND

NEWFOUNDLAND

Dr. N. S. Fraser, dean of the medical profession in St. John's, recently entered his 90th year and his 67th year in medical practice. On the occasion of these special anniversaries Dr. Fraser received the congratulations of his many friends and at the last monthly meeting of the St. John's Clinical Society he was accepted as the first honorary member of that Society.

Dr. Fraser was educated at the Collegiate Institute, Kingston, Ont., and matriculated for Queen's University winning the MacKerras' Memorial Scholarship in Latin and Greek. At Edinburgh University he was in the honours class. Dr. Fraser (N.S.F. to many readers) is also well known as a contributor of timely letters and articles to the press of St. John's.

Interesting programs were recently held under the sponsorship of the St. John's Clinical Society. At a meeting held in the St. John's Sanatorium Drs. A. G. Jessamine and W. D. Heneghan delivered excellent papers on chemotherapy and collapse measures in the modern treatment of pulmonary tuberculosis. A comprehensive talk on "Strabismus in Children" was presented by Dr. J. H. Malloy at a session held in the General Hospital.

Drs. P. J. Whelan of St. John's, R. L. Lawton of Millertown, and R. F. O'Driscoll of Grand Falls attended refresher courses at Dalhousie University, Halifax, during the month of April.

Dr. George Flight, now practicing in Channel, will be leaving shortly for Halifax to take up postgraduate studies in Obstetrics and Gynæcology at the Victoria General Hospital.

Drs. Abe Levitz and Agnus Neary recently returned to their homes in St. John's to spend a short vacation. Dr. Levitz is attached to the Staff, Department of Anæsthesia, Royal Victoria Hospital, Montreal. Dr. Neary is doing postgraduate studies in Surgery at the Mayo Clinic. P. F. ASHLEY

ONTARIO

Dr. Abram I. Willinsky gave an address entitled "Stupor Mundi", a study of some aspects of medicine in the 13th century, at the Library and Historical night of the Toronto Academy of Medicine.

A hobby show of the fellows was displayed at the Academy. Paintings led the exhibit in numbers. Photography was well represented with many beautiful colour films. There were exquisite wood carvings, done by a surgeon, collections of semi-precious stones, collections of sea shells and handmade furniture. Several examples of fly-tying by fishermen members were shown.

Dr. T. G. H. Drake exhibited rare items from his collection of medical curios, including surgical tools, amulets and ancient feeding bottles. Dr. James Goodwin displayed rare Canadian stamps and other interesting Canadiana.

The provincial welfare department conducted a week's course at the Ontario County Home for superintendents and staff members of homes for the aged. About twenty-four representatives from all over the province attended. Topics discussed included business administration, general care of the aged, the psychology of the older person, recreation, occupational therapy, fire safety, nutrition and public relations.

Samuel I. Yamada has been awarded a fellowship valued at $2,000 for heart disease research by the Life Insurance Research Fund. He will continue his work under Dr. A. G. Burton on biophysics at University of Western Ontario.

New Quarters for the Toronto Branch and the Ontario Division of the Canadian Cancer Society are at 276 Simcoe St. where the work of the Cancer Dressing Station is carried on. The Gift Cupboard has room to expand and there is an auditorium. There is also a room for the Laryngectomy speech classes and "The Lost Chord Club".

Physicians' Services Incorporated has reported that in 1952 the enrolment was 320,068 participants and 3,650 participating physicians. Among this group of patients there were 5,300 tonsillectomies, 4,700 confinements, 2,700 fractures and 1,600 appendectomies.

Sir Thomas Dunhill, who attended as senior surgeon, King George VI during his most serious illnesses, paid a visit to Dr. Robert Janes recently. Sir Thomas, now 76, on his way home from a visit to his native Australia, with stops at Cleveland and St. Louis, where he visited surgical friends was looking forward to the fishing at his country home in England.

Canad. M. A. J.
June 1953, vol. 68

Dr. Paul M. St. Aubin, a graduate of University of Toronto, has been awarded the Allan Blair Memorial Fellowship valued at $8,000. It provides two years of advanced professional training for a Canadian medical graduate specializing in clinical work related to the diagnosis and treatment of cancer.

Dr. J. Harris McPhedran, Toronto was elected president of the College of Physicians and Surgeons of Ontario at the 88th annual meeting. Dr. James A. Dauphinee, Toronto was elected vice-president.

Other officials are: executive committee: Dr. J. H. McPhedran, Dr. J. A. Dauphinee, Dr. Carl E. Hill, Lansing; Dr. J. F. Sparks, Kingston; Dr. Ward Woolner, Ayr.

Discipline committee; Dr. D. S. Wigle, Windsor, Dr. M. H. V. Cameron, Toronto; Dr. J. C. Gillie, Fort William; Dr. A. L. Richard, Ottawa; Dr. A. B. Whytock, Niagara Falls.

Education and Registration Committee; Dr. Malcolm Brown, Kingston; Dr. J. F. Argue, Ottawa; Dr. F. S. Brien, London; Dr. Malcolm Brown; Dr. G. C. Jarrott, Stratford; Dr. D. S. Wigle; Dr. A. B. Whytock.

Finance, Printing and Property Committee; Dr. Carl E. Hill; Dr. M. H. V. Cameron; Dr. G. C. Jarrott; Dr. R. W. Schnarr, Kitchener; Dr. Ward Woolner.

The Alcoholism Research Foundation and Brookside Clinic, with new headquarters at 9 and 11 Bedford Road, Toronto were recently opened by Health Minister Phillips. On the staff headed by H. David Archibald, executive director and Dr. John D. Armstrong, medical director, are nurses, a receptionist, a social worker Mrs. Marion Hummel and a psychiatric social worker, Miss Margaret Cork. There is one male nurse. Ten beds in the hospital at Bedford Road are for men and two for women patients. The foundation co-operates with doctors in other places and 25% of patients have come from outside Toronto. In the past two years 1,100 patients have been cared for by the foundation.

During 1952 there were 19,908 people or 12% of those between 60 and 69 years in Ontario receiving old age assistance. The Old Age Assistance Act was adopted to take care of persons in the 65-69 year age group who could qualify for assistance under a means test. This assistance program is financed on a 50-50 basis by provincial and federal authorities, up to a basic figure of $40 a month.

Under the Act, assistance is available to those whose income, including assistance, is not more than $720 a year for an unmarried person and $1,200 for a married person.

Dr. W. T. Hamilton, professor of Anatomy, Charing Cross Hospital Medical School, London, lectured at University of Toronto on April 20 and 21. His subjects were; New Concepts of the Placenta and The Placenta and Abnormal Development. LILLIAN A. CHASE

QUEBEC

On Thursday, April 16, starting at 7.30 p.m., a group of staff doctors of Queen Mary Veterans' Hospital, Montreal, put on an exhibition evening for the members of the St. Francis Valley Medical Society at Sherbrooke, Quebec. Fifty-six members of the Society were present at the meeting, including the president, Dr. W. Klinck of Lennoxville, and the Secretary-treasurer, Dr. J. MacLeod of Sherbrooke. The meeting was held at St. Vincent de Paul Hospital and all arrangements for facilities there as well as for refreshments were made by Dr. Jacques Olivier of Sherbrooke. The D.V.A. part of the program was arranged by Dr. R. G. Townsend, Consultant in Orthopædics, and Miss Eleanor Sweezey, Director, Department of Medical Illustration. This was a very enjoyable and profitable evening, and it would seem worthwhile to have more joint meetings of this type.

Sir Sydney Smith, F.R.S., C.B.E., Dean of Medicine of Edinburgh University, delivered on May 1 the inaugural Shepherd Memorial Lecture under the auspices of the Montreal General Hospital at Moyse Hall, McGill University. This lecture was in memory of Dr. Francis Shepherd who was on the visiting staff and later chief surgeon and consultant at the General Hospital from 1883 until his death in 1929. He was also Dean of the Faculty of Medicine at McGill from 1908 to 1914.

Sir Sydney was introduced to the large gathering by Dr. E. S. Mills, Chairman of the Hospital Medical Board, while Dr. Alfred T. Bazin gave a brief address dealing with the life and achievements of Dr. Shepherd. Dr. C. A. Peters thanked the lecturer and presented him with a mantel clock designed by Mr. Percy Nobbs, son-in-law of Dr. Shepherd and former Professor of Architecture at McGill. The face of the clock is a replica of the hospital seal.

In turn Sir Sydney presented to Dr. Mills for the hospital a number of historical documents including indentures for surgery apprentices dated 1653 and 1727, a prescription written by Lister in 1892, a case book of James Syme, famed surgeon of the pre-Lister period, dated 1841, and a citation when Dr. Shepherd was given an honorary degree at Edinburgh.

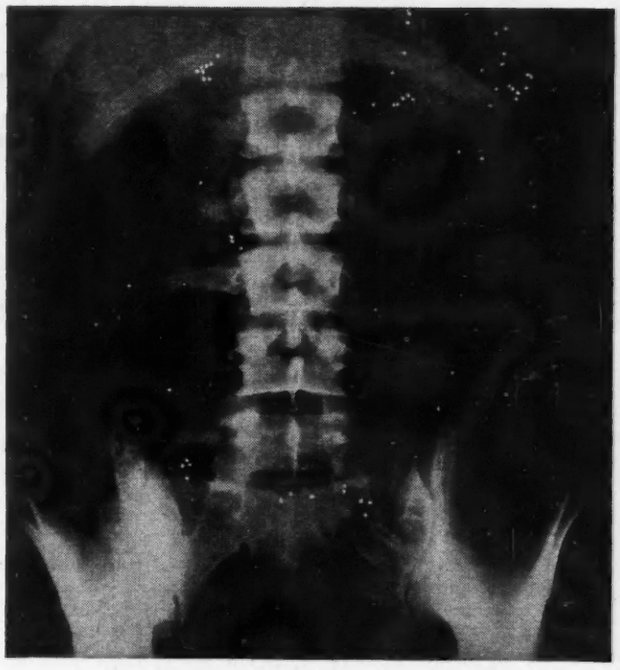
The cornerstone of the new Montreal General Hospital was laid on May 2 by Premier Maurice Duplessis. This nineteen-storey building, between Pine and Cedar Avenues just east of Cote des Neiges and on the slopes of Mount Royal, will, when completed, replace the obsolete Central Division on Dorchester Street East and the Western Division at Atwater Ave. The latter division will be taken over for the new Children's Memorial Hospital.

Premier Duplessis used a silver trowel loaned for the occasion by Edinburgh University whose Dean of Medicine, Sir Sydney Smith, attended the ceremony. This trowel had performed a similar function in 1897 when Lord Lister laid the foundation stone of the former Nurses' Home of the Montreal General on Dorchester Street. It has since been in the museum of the Scottish university.

The new cornerstone of this hospital contains microfilms of the contents of the Richardson Wing cornerstone laid in 1832 on Dorchester Street, copies of the annual reports of the hospital for 1821 and 1952, a set of coins in current use, program and photograph of the present dedication ceremony, photostats of teaching cards issued to students in 1821, first list of diseases issued by the hospital the same year, a brief history of the Joint Hospital Fund Campaign in 1950 and plans for the forthcoming campaign this month, and a copy of the May, 1953, issue of the *Hospital Bulletin*.

The eighteenth annual Hughlings Jackson Memorial Lecture at the Montreal Neurological Institute will be given this year by Dr. James C. White, Assistant Professor of Surgery, Harvard Medical School, and Chief of the Neurosurgical Service of Massachusetts General Hospital. Dr. White will speak on "Pain Conduction in Man: Studies on its Transmission in Spinal Cord and Visceral Plexuses". The lecture will be given at the Institute on May 13 at 5 p.m.

Two new appointments in the Faculty of Medicine of McGill University have recently been announced, both to take effect as of July 1. Dr. Donald R. Webster has been appointed as Surgeon-in-Chief at the Royal Victoria Hospital and Professor of Surgery in the Faculty of Medicine. Dr. Webster will succeed Dr. G. Gavin Miller who will relinquish both positions in order to devote his full time to private practice. Dr. Alan S. Ross has been appointed Physician-in-Chief of the Children's Memorial Hospital and Professor of Pædiatrics



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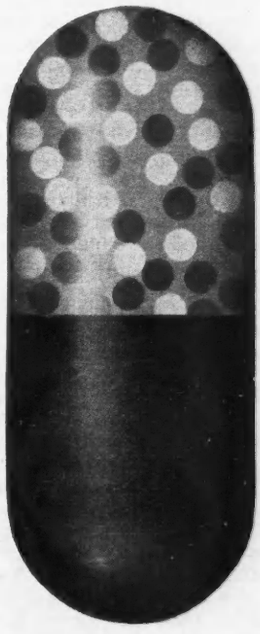

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Canad. M. A. J.
June 1953, vol. 68

at the University. Dr. Ross will succeed Dr. Alton Goldbloom who will relinquish both positions also in order to devote his full time to private practice.

In announcing these full-time appointments, McGill University and hospital authorities state that experience has proved that such an arrangement, while allowing appropriate opportunities for private practice, is of great assistance in regard to the tasks of administering hospital services and supervising the education of medical students.

A. H. NEUFELD

SASKATCHEWAN

Dean J. Wendell Macleod of the University of Saskatchewan was the guest of honour at a special banquet held in Moose Jaw on March 16, 1953. His topic was "Medical Education".

A new hospital is to be built at Eston, Sask., to replace the one destroyed by fire last December. It will serve about 3,500 people and will have 25 beds, a six bassinet nursery; medical, obstetrical, x-ray and laboratory facilities and a community health centre. The federal and provincial governments are each contributing $24,000.00 toward the building costs.

A $6,000.00 grant has been given to the Assiniboia Union Hospital where alterations are to be made this year to increase the hospital's bed capacity by six.

An important addition to the Medical Library of the University of Saskatchewan will be The John R. Brodie Collection of Medical History and Humanities. Last Fall the Board of Governors of the University acknowledged receipt of a gift of $1,000 from Mr. John R. Brodie of Winnipeg to lay the basis for a collection of volumes dealing with the historical background of medicine and its relations to other cultural and academic disciplines. Mr. Brodie, who in boyhood in Montreal was both friend and admirer of the late Dr. William H. Drummond, family physician and poet, expressed the hope that this collection would stimulate students to further interest in the great men who combined in their lives the practice of good medicine, articulate literary taste and an absorbing interest in humanity. This goal is in line with current trends in medical education and the Brodie Collection is expected to serve as an important reference aid to both students and staff.

In a series of lectures, class conferences and small group discussions, Dean Wendell Macleod and staff will present an orientation course for first year medical students entitled Introduction to Medicine. In the University calendar its content is described as "The heritage of medicine; its relation to the evolution of science, society and the humanities. Health customs and conceptions of disease as human reactions to scientific, socio-economic and cultural settings. Techniques and ethics of medical practice from origins to present patterns. Points of departure for study of preventive medicine and public health, statistical methods, interviewing and the behaviour of individuals and groups." Valuable guidance in the selection of books for the John R. Brodie Collection has been received from Dr. Erwin H. Ackerknecht, Professor of History of Medicine, University of Wisconsin; Dr. W. W. Francis, The Osler Library, McGill University; Dr. John F. Fulton, Professor of History of Medicine, Yale University; Dr. Ralph Major, University of Kansas; and Dr. Earle P. Scarlett of Calgary, Chancellor of the University of Alberta. Gifts of books have been received from Dr. Francis and from the library of the late Dr. H. P. Wright of Montreal.

Following attendance at the meeting of the Medical Librarians' Association in Salt Lake City in June, Miss Grace Giles will visit a number of Western medical libraries. Her penchant for second-hand book stores is expected to lead to some interesting acquisitions by the Saskatoon library.

W. PEACOCK

NEWS OF THE MEDICAL SERVICES

Canadian Armed Forces

During the annual DGMS Tactical Exercise held at Camp Borden between March 24-28, 1953, one day was devoted to a consideration of the surgical problems met in Korea. A panel of surgeons and anæsthetists, under the chairmanship of Col. J. E. Andrew, discussed the management of burns, chest wound, advances in vascular surgery and the use of the artificial kidney in a theatre of war.

The Professional Advisory Staff of the DGMS met in Camp Borden on March 28, 1953 following the annual Tactical Exercise. Numerous problems relating to professional standards and professional training of medical officers of the army were discussed.

Group Captain DGM Nelson, Deputy Director Medical Services (Air), and a representative group of Medical Officers from the RCAF attended the 24th Annual Meeting of the Aero Medical Association held in Los Angeles California May 11 to 14, 1953. Scientific papers in the field of Aviation Medicine were presented by those officers attending.

A representative from the Directorate of Medical Services (Air) attended the Biennial Meeting of the Canadian Hospital Council held in the Chateau Laurier, Ottawa, May 18 to 20, 1953.

NEWS AND NOTES

Grants and fellowships in the field of heart disease research totalling more than $810,000 have been announced in New York by the Life Insurance Medical Research Fund, an organization of United States and Canadian life insurance companies. The new awards raised to more than five and one-half million dollars the money which has been contributed to research by the Fund since it was organized late in 1945.

Since the Fund began operation, it has supported 205 research programs and 241 research fellowships and its work has been carried in 98 different institutions. Numerous important additions to knowledge about heart disease have been made and many important leads will be investigated during the coming year directed to better methods of treating heart disease and obtaining more knowledge about how it may be controlled or prevented. In the elections held to choose new members of the Fund's board of directors, E. C. Gill, President of the Canada Life Assurance Company, Toronto, was among those named.

Grants to Canadian institutions in aid of research on diseases of the heart and arteries included:

Hotel-Dieu Hospital, Montreal, Quebec, for research by Dr. Jacques Genest, on the relationship of hypertension in man to the sodium-retaining factor of the adrenals.

University of Saskatchewan School of Medical Sciences, Saskatoon, Saskatchewan, for research by Dr. L. B. Jaques, on the physiology of heparin and its relation to thrombosis.

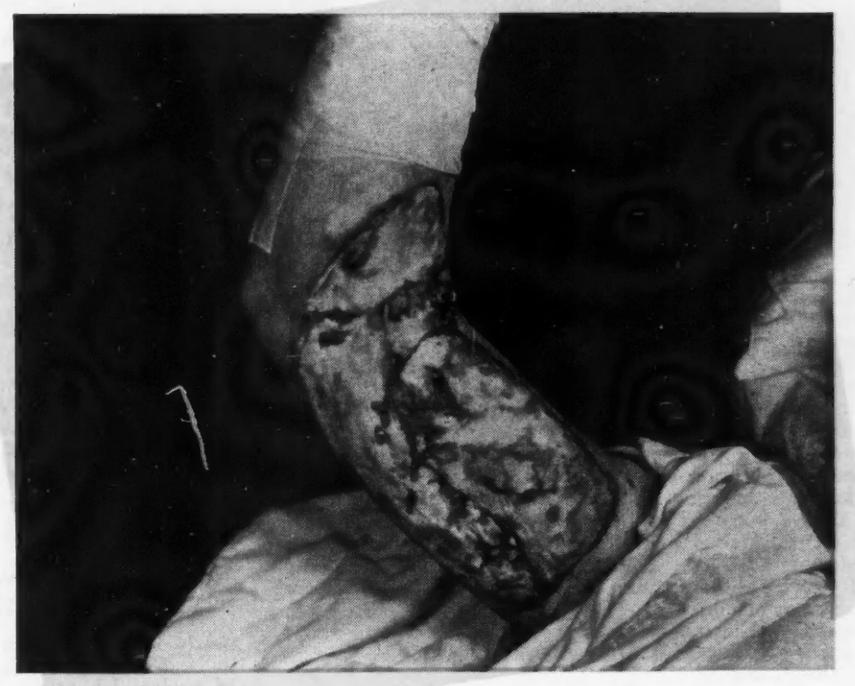
University of Western Ontario Faculty of Medicine, London, Ontario, for research by Dr. Alan C. Burton, on the hæmodynamics of small blood vessels.

Canadians awards by the Fund for Postdoctoral Research Fellowships include:

R. M. Cherniack, M.D., of Winnipeg, Man., for research under the guidance of Dr. Richard L. Riley, Johns Hopkins University School of Hygiene and public Health, Baltimore, M.D.

Peter Gaskell, M.D., of London, Ontario, for research under the guidance of Professor Henry Barcroft, Sherrington School of Physiology, St. Thomas' Hospital, London, England.

(Continued on page 60 of the advertising section)



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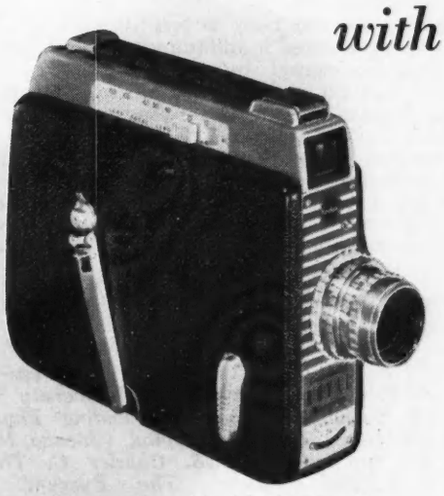

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Canad. M. A. J.
June 1953, vol. 68

BOOK REVIEWS

THE KNEE

P. Lewin, Professor and Chairman of Department of Bone and Joint Surgery, Northwestern University Medical School; Attending Orthopædic Surgeon, Cook County Hospital. 914 pp. illust. $17.60. Lea & Febiger, Philadelphia; The Macmillan Company of Canada Limited, Toronto, 1952.

The author must be congratulated on this remarkable book. It would not be wise to discuss each of the 44 chapters. Some are very short, and one may question the merit of their individuality. However, one sees the author's continuous attempt at clarity in organizing this book as he did. The first 13 chapters deal with the structure and mechanics of the knee, and the problems of diagnosis. The rest of the volume is dedicated to a careful discussion of any subject that may possibly be related to the knee. The anatomy of the specific lesions is always discussed, and is followed by a complete review of all the known methods of therapy. The illustrations are most adequate. The boxing of the tables is equally effective. The long list of references allows the reader a ready opportunity to study the original literature with ease. To those interested in reading and learning about the knee and related structures, Dr. Lewin's book is to be recommended.

THE SCALP IN HEALTH AND DISEASE

H. T. Behrman, Assistant Clinical Professor Dermatology New York University Post-Graduate Medical School; Adjunct Dermatologist, Mount Sinai Hospital; Attending Dermatologist, Hillside Psychiatric Institute. 566 pp. illust. $14.00. C. V. Mosby Co., St. Louis; McAinsh & Co. Ltd., Toronto, 1952.

This text, confined in its scope to the consideration of the scalp and hair, appears to cover most of the known facts of this very expensive part of our anatomy. When one considers the amount of money spent each year by people, particularly the female sex, upon healthy scalp it almost staggers one's imagination at the total cost of normal and diseased conditions.

In this work the various methods of cutting, bleaching, dyeing, curling or waving are carefully explained; not to mention the effort to destroy the hair or make it grow. The approach to all these discussions and treatments is a logical one since it begins with the formation, then the anatomy of the hair and scalp along with its intricate glandular and circulatory system. The second chapter explains adequately the various hair preparations as used to-day on normal individuals. The various diseased conditions of the scalp and hair are then most fully considered; and I know of no work which is so complete. The formulary covers several pages and should satisfy anyone since the prescriptions are simple yet well chosen. The illustrations of disease are excellent and not as in some texts of rarities; but they depict the common or average type of case.

Dermatologists cannot be without this work and practitioners of medicine will find it a very valuable addition to their library if only to answer with assurance the many questions put to them about the scalp and hair in health and disease.

SURGICAL CARE

R. W. Raven, Officer of the Order of St. John of Jerusalem; Chevalier de la Légion d'Honneur; Honorary Colonel, Royal Army Medical Corps. 422 pp. illust. 2nd ed. $7.50. Butterworth & Co. (Canada) Ltd., Toronto, 1952.

This volume outlines the pre- and post-operative care of surgical patients. Shock, hæmorrhage, protein deficiency, and water and electrolyte balance are discussed in the opening chapters. Then follow instructions for the care of patients undergoing almost, if not all, types of surgical treatment. Instructions are clear, concise, dogmatic, expressed in the light of the author's experience. Many points undoubtedly vary from the procedure of others. On few points will one be opposed, but the statement that oxygen therapy is not of value in the treatment of burns is a statement contrary to the reviewer's experience. The book will be of interest to surgeons, and of value to postgraduate students in both surgery and nursing.

TROPICAL MEDICINE

L. Rogers, Major-General, Indian Medical Service, Ret.; late Medical Adviser to the India Office and J. W. D. Megaw, Major-General, Indian Medical Service, Ret.; Late Medical Adviser, Indian Office and Lecturer, London School of Tropical Medicine. 560 pp. illust. 6th ed. $8.00. J. & A. Churchill Ltd., London; British Book Service (Canada) Ltd., Toronto, 1952.

The numerous discoveries of the last war made considerable changes necessary which have resulted in an increase in the size of the present edition. As before, the needs of medical students and of practitioners who work in conditions in which they cannot utilize the facilities of modern laboratories, have been kept in mind. Special stress has been laid on the methods of diagnosis, which can be carried out with the help of a microscope and a few simple appliances. The newer methods of treatment, such as the antibiotics, have been included, but at the same time reference is also made to older methods of treatment, in view of the price of some of the new drugs, which makes their use sometimes impossible in backward communities.

On the whole, the book covers diseases which are likely to be encountered in the tropics, protozoal diseases, febrile diseases caused by spirochætes, by filtrable viruses and bacteria, and rickettsial diseases. Chapters on diarrhœal diseases, conditions affecting the body surface, helminthic diseases, dietary deficiency diseases and lesions caused by heat and light and by venomous animals are included.

There are a number of useful illustrations and diagrams. The sections are concise and are written by eminent experts with a vast experience in their field.

ESSENTIALS OF INFANT FEEDING FOR PHYSICIANS

H. F. Meyer, Assistant Professor, Department of Pædiatrics, Northwestern University School of Medicine, Associate Attending Physician, Children's Memorial Hospital, Chicago, Illinois. 252 pp. illust. $9.25. Charles C. Thomas, Springfield, Illinois; The Ryerson Press, Toronto, 1952.

Dr. Meyer has produced a readable and interesting text on a subject which has shown no startling changes in recent years. It is not too technical and will be useful to the man in general practice. Breast feeding is fully discussed. Reasons for failure are simple and to the point. Sound advice is given as to the methods that are useful in helping the prospective mother both before and after her baby is born. The chemical aspects are well outlined. This chapter is clear and instructive. Artificial feeding is divided into mechanics, construction of mixtures and there is a lengthy chapter on proprietary foods. This is helpful in view of the large number of such products in use. His advice is to limit the number of preparations used and to be thoroughly acquainted with them. He regrets the duplication of so many of these foods. Solid food supplements and vitamins are fully discussed and the final chapters cover psychological feeding problems, self-demand and rooming-in.

Canad. M. A. J.
June 1953, vol. 68

EMERGENCIES IN MEDICAL PRACTICE

Edited by C. A. Birch, Physician, Chase Farm Hospital, Enfield. 587 pp. illust. 3rd ed. $6.25. E. & S. Livingstone Ltd., Edinburgh and London; The Macmillan Company of Canada Ltd., Toronto, 1952.

One is amazed to find that there are so many emergencies in medical practice. A glance at the table of contents shows that emergencies are discussed in all the various systems of the body as well as particular emergencies at sea, in the air, in the tropics, in industry, various poisons, etc. The list is an imposing one and the problem is discussed in a clear and easy, readable and practical manner. Many of the problems require resort to a textbook as it is the type of information that one does not frequently need, and hence is not easily remembered. This text is so conveniently written that it should certainly find a place in the medical practitioner's library, if not in his medical bag. The author has written many of the chapters himself, and a very interesting and informative one on "Medical-legal and other Non-Clinical Emergencies". In addition there are chapters written by 21 other specialists who deal specifically with emergencies in their own respective fields in an authorative manner.

THE SKULL AND BRAIN ROENTGENOLOGICALLY CONSIDERED

C. W. Schwartz, Associate Professor of Clinical Radiology, College of Physicians and Surgeons, Columbia University, and L. C. Collins, Associate Professor of Radiology, College of Physicians and Surgeons, Columbia University. 386 pp. illust. $12.50. Charles C. Thomas, Springfield, Illinois; The Ryerson Press, Toronto, 1951.

The authors' foreword to this volume states it "was compiled for the sole purpose of producing a source of ready reference for those who may be called upon to examine roentgenograms of the skull". This purpose is well achieved, using 335 figures which are almost uniformly of very high quality reflecting credit upon radiographer and publisher alike. The length of the text has been sacrificed to the illustrations, but contains much useful information with emphasis on the sections dealing with skull and intra-cranial tumours and miscellaneous diseases. The roentgenological consideration of the brain indicated in the title does not include cerebral angiography, ventriculography and pneumoencephalography.

The book, with the key references included at the end of each chapter, fills a distinct need for neurologist and neurosurgeon and for radiologists generally. It is recommended as the starting point for investigation of the perplexing skull film.

THE STORY OF ST. LUKE'S HOSPITAL

C. N. French. 212 pp. illust. $2.00. William Heinemann Ltd., London; British Book Service (Canada) Ltd., Toronto, 1951.

This small book recalls the ever absorbing history of the treatment of insanity. St. Luke's Hospital, like many of its contemporaries, grew out of the humane impulses of a small group of men. In 1750 six London gentlemen met to consider the establishment of a hospital for the care and treatment of poor lunatics. Any such plan had to deal with inertia, with prejudice and even with a desire for entertainment, as it was an accepted social pleasure to go and look at the lunatics. One of the first rules of St. Luke's was to forbid exposing patients to public view, and this in itself is sufficiently impressive evidence of the horrible conditions of the time.

Charles Dickens' reprinted account of a visit to the hospital on the night of Boxing Day in 1851 is a minor history of the treatment of the insane, written with all the poignancy which such terrible things always evoked in Dickens. It is used in the book to show how much even in the 50 years after his visit the conditions had improved. The book well recalls the humanitarian spirit of this fine institution.

Books Received

Books are acknowledged as received, but in some cases reviews will also be made in later issues.

Operative Neurosurgery. E. S. Gurdjian, Professor of Neurosurgery, Wayne University College of Medicine; Chief, University Neurological Service, Grace Hospital, Detroit, Michigan; and J. E. Webster, Assistant Professor of Surgery, Wayne University College of Medicine; University Neurosurgical Service, Grace Hospital, Detroit, Michigan. 422 pp., illust. $11.00. The Williams & Wilkins Company, Baltimore; Burns & MacEachern, Toronto, 1952.

Gynecological and Obstetrical Pathology. P. A. Herbut, Professor of Pathology, Jefferson Medical College and Director of Clinical Laboratories, Jefferson Medical College Hospital, Philadelphia, Pennsylvania. 683 pp., illust. $13.75. Lea & Febiger, Philadelphia; The Macmillan Co. of Canada, Toronto, 1953.

Applied Microbiology. Published under the Sponsorship of The Society of American Bacteriologists. Vol. No. 1. It is issued bi-monthly and subscription is $7.50. The Williams & Wilkins Company, Baltimore, Maryland, 1953.

Les Vomissements du Nourrisson. E. Roviralta, Chef du service de chirurgie infantile et orthopédique de l'Institut Policlinique de Barcelone. 236 pp., illust. Prix: 1,800 francs. Editions Médicales Flammarion, Paris, 1952.

The Biochemistry of Gastric Acid Secretion. E. J. Conway, Professor of Biochemistry and Pharmacology, University College, Dublin, Ireland; Honorary Fellow, Royal College of Physicians, Ireland. 185 pp., illust. $7.75. Charles C. Thomas, Springfield, Illinois; The Ryerson Press, Toronto, 1953.

Pediatrie. Collection Médico-Chirurgicale à Révision Annuelle. R. Debré et M. Lelong. Deux volumes reliés, 2,260 pp. Figures. Les 2 volumes 14,500 Frs. Les Editions Médicales Flammarion, 1952.

Pathology in Surgery. E. F. Hirsch, Director of the Henry Baird Favill Laboratory and Pathologist of St. Luke's Hospital, Chicago, Illinois. 474 pp., illust. $11.00. The Williams & Wilkins Company, Baltimore; Burns & MacEachern, Toronto, 1953

Cancer in General Practice. R. W. Raven, Officer of the Order of St. John of Jerusalem; Chevalier de la Légion d'Honneur; and P. E. T. Hancock, Physician, The Royal Free Hospital. 265 pp., illust. Butterworth & Co. (Canada) Ltd., Toronto, 1952.

JOURNAL OF

Canadian Medical Association

Editorial offices — 3640 University St., Montreal 2
General Secretary's office—135 St. Clair Ave. W., Toronto

Subscription rates: The Journal is supplied only to paid up members of the Canadian Medical Association with the following exceptions: for medical libraries, hospitals and doctors residing outside of Canada, the annual subscription is $10.00; for medical students residing in Canada there is a special rate of $2.50 per annum. All subscriptions and related correspondence should be addressed to the General Secretary's office at 135 St. Clair Avenue West, Toronto 5, Ontario.

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Papers should be kept below 4,000 words wherever possible. Whilst not necessarily a cause for rejection, excessive length of an article is undesirable.

References: in the case of a journal arrange as follows: author (JONES, A. B.), title, journal, volume, page, year. In the case of a book: WILSON, A., Practice of Medicine, Macmillan, London, 1st ed., p. 120, 1922.

Illustrations: A limited number will be accepted. Photographs should be clear: drawings should be in india ink on white paper. All unmounted. Legends to be typed separately.

Reprints: May be ordered upon forms sent with galley proofs.

News: The Editor will be glad to consider any items of news that may be sent in by readers.

CLASSIFIED ADVERTISEMENTS

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NOTICE.—PUBLIC HEALTH. For information about full-time appointments as County Health Units and Medical Officers, or about fellowships for postgraduate training (Diploma in Public Health), address the Executive Secretary, Canadian Public Health Association, 150 College Street, Toronto 5, Ontario.

NOTICE.—PRACTICE IN WINNIPEG. Medical doctor to share new building with dentist. Office space ideally laid out. Excellent location in residential-business section with 10,000 population in immediate neighbourhood. Only one other physician in district. Apply to Box 634, Canadian Medical Association Journal, 3640 University Street, Montreal.

NOTICE.—INTERNSHIP. Rotating service comprises: 1. Surgery and Emergency; 2. Medicine and Pædiatrics; 3. Pathology; 4. X-Ray; 5. Obstetrics; (3, 4, and 5 optional). Because this is a hospital of approximately 200 beds, there is a satisfactory relationship between the staff and the interns. A special effort will be made to see that the interns get good work and basic training with a maximum of interest. Remuneration: $50.00 to $100.00 depending upon qualifications. Applications to be made to: The Superintendent, St. Joseph's General Hospital, Port Arthur, Ontario.

NOTICE.—Approved internships now available at Marymount Hospital located in Garfield Heights, a suburb of Cleveland, Ohio. 200-bed capacity. Approved residencies in surgery, medicine and anæsthesiology. Stipend $200.00 a month less $50.00 for board. Apply to: Administrator, Marymount Hospital, 12300 McCracken Road, Garfield Heights 25, Ohio, U.S.A.

FOR SALE.—Office suite and living quarters in the business district of a south western Ontario village. Large unopposed practice waiting. Eight miles from a hospital. Excellent start for intern ready for general practice. Terms arranged. Apply to Box 566, Canadian Medical Association Journal, 3640 University Street, Montreal.

FOR SALE.—Vancouver, B.C. Well-kept residence and office combined for physician-surgeon. Last year's income exceeded $20,000, and could be increased easily. Hospital affiliation can be readily arranged. Amount of work depends entirely on the man. Property valued at $20,000, half of which may be carried on mortgage at 5%, if so desired. Reply to Box 633, Canadian Medical Association Journal, 3640 University Street, Montreal.

FOR RENT.—Owing to recent death of general practitioner, fully equipped office has become vacant for rent; also position for health officer now open. Living quarters available. Apply to: Mrs. M. G. Ranney, Kinmount, Ontario.

FOR RENT.—Office for young certified internist, waiting-room shared with certified general surgeon, in Guelph, Ontario. Office ideally situated in this growing city. Rent reasonable. Two hospitals in city, with excellent x-ray and laboratory facilities. Beds readily available. Apply to Box 636, Canadian Medical Association Journal, 3640 University Street, Montreal.

FOR SALE.—Lucrative practice in Alberta. Net returns of practice are above national gross. The payment includes all equipment (worth $9,000). Price: one-third of yearly net. Good accommodation. Terms can be arranged, if required. Apply to Box 655, Canadian Medical Association Journal, 3640 University Street, Montreal.

FOR SALE.—Lucrative general practice in village in Southern Saskatchewan, income guaranteed by prepaid scheme, fully equipped and modern hospital in the village, with fully trained staff. Two-storey solid brick clinic and residence, fully modern with assistant's living quarters in separate building on same grounds. Will sell immediately for value of real estate. Amount involved about half of yearly income. Owner leaving for post-graduate work. Apply to Box 654, Canadian Medical Association Journal, 3640 University Street, Montreal.

FOR SALE OR RENT.—Doctor's office in Toronto for sale or rent. Valuable corner property. Well established 20 year-old practice. Forced to sell because of illness. Building suitable for subdivision into several professional suites or clinic. Present office large, ultramodern, fully equipped including X-ray. EM. 6 - 9226.

Continued on Page 36

Thirty Years Ago . . .

From the *Journal* of June, 1923

PROBLEMS OF OBSTETRICAL PRACTICE–

W. W. Chipman

"Parturition is a physiological process, identical in the countess and in the cow. At least, until recently, it has been so regarded. Physiological the process is, and yet as regards the countess–and every mother is a countess–this natural process still remains a hazardous occupation. The price of motherhood is still 'cruel high': ask any insurance company.

"If it is important to be in the world at all, the matter of this entrance, and the price of admission, are surely of first consideration. And yet, oddly enough, as compared with the sister subjects of medicine and surgery, obstetrics has been, not a first, but a last consideration, and it still runs against these a bad third in the race. . . . .

"Our special service is to provide, as near as may be, a universal Yea, to the mother and her child: 'Is it well with the child?' And, by reason of our service, the mother herself may make answer, 'With myself and my child, it is well.'"

♧ ♧ ♧

"The Annual Report of the Saint John County Hospital is just at hand, and proves a revelation to those who have not been familiar with the work done there. Under Dr. H. A. Farris the institution has become one of the most efficient and best equipped in Canada. The addition of an operating room where surgery of the thorax may be done, is a much needed improvement and was the one thing needed to bring the hospital thoroughly up-to-date.

"It was with great regret that the profession learned that Dr. Stewart Skinner, for many years Registrar for New Brunswick, had found it necessary, on account of his health, to resign. Fortunately, his successor, Dr. J. S. Bentley, has the goodwill of all the physicians in the province, and will prove a worthy official. His position is not a sinecure, but his popularity with his professional brethren will make the work less irksome than it otherwise would be."

♧ ♧ ♧

NEWS ITEMS–Ontario

MEDICAL LEGISLATION

"The Legislative Assembly of the Province of Ontario passed the third reading of the Medical Bill on Friday, May 4, 1923. Although the Bill, which is herewith appended, does not compel the present irregulars in the province to cease practising, it does aim to accomplish three great things, namely, (1) defining the 'Practice of Medicine'; (2) demanding that, in the future, all who desire to practice in this province must be educated; (3) placing prosecutions where they belong, namely, in the hands of the County Crown Attorneys. There is much cause for gratification on the part of the medical profession of the province."

♧ ♧ ♧

EVERY DAY AND EVERY WAY

"Oh Mr. Coué comes from France and from the town of Nancy,
His 'every day and every way' caught many persons' fancy.
His views were put into a book, 'tis found in many places,
'Imagination rules the will'–its keynote and its basis. . . .

"What need for ointments or for pills, for this or that fine potion?
Away with salts! confound your squills! no more of any lotion!
And why seek constipation's cure, by lengths of bowel resected?
When all may learn the 'day and way' a lesson resurrected. . .

"The views which Coué has advanced are found in many pages,
From Charcot back to Plato's time, and in the Middle Ages,
That well they knew the mind of man can be now predicated,
But then their views did not bring cash, nor were they syndicated."

E. J. Mullally

NEWS AND NOTES

(Continued from page 638)

Mary Louise McFadden, M.A., of Vancouver, B.C., for research under the guidance of Dr. Emil L. Smith, University of Utah, Salt Lake City, Utah.

Samuel I. Yamada, M.Sc., of Toronto, Ont., for research under the guidance of Dr. A. C. Burton, University of Western Ontario, London, Ont.

U.N. COMMISSION ON NARCOTIC DRUGS

K. C. Hossick, chief of the federal health department's narcotic control division, has been appointed as alternate delegate to the eighth session of the United Nations' Commission on Narcotic Drugs. This commission, which meets yearly, is the policy-making body on all questions relating to narcotic control and advises the Economic and Social Council of the United Nations on the problems of applying and supervising the application of the various international agreements for the control of narcotic drugs. Canada is one of the 10 permanent members of the commission. Canadian delegate to the commission since it was formed in 1946 is C. H. L. Sharman, Ottawa.

Winter's added threat to life is much less marked than it was forty years ago, according to the Metropolitan Life Insurance Company's statisticians. A study of the monthly variation in deaths shows the winter peak in the mortality curve to have flattened noticeably since the early years of this century. The change is attributed in large measure to the remarkable progress made in the control of pneumonia and other respiratory infections.

"The levelling has not been as evident at the older ages as in other periods of life," the statisticians observe, "primarily because respiratory complications still contribute to the death of many older persons with heart and other degenerative diseases. However, even at ages 60 and over, the death rate is not as responsive to seasonal variations as it was in former years."

The current situation among the very young is in sharp contrast to that of a century ago, when the death rate, particularly in densely populated areas, was highest during the summer months because of major outbreaks of diarrhœa and other intestinal diseases. With improvement in milk and water supplies, garbage disposal, and sanitary conditions in general, the summer peak gradually subsided.

Even after the turn of the century, the statisticians point out, infants and young children still succumbed in largest numbers during the summer. In 1907-1911, the death rate in New York State for children under five years of age was two-fifths higher in August than the average for the year as a whole. By contrast, currently the summer months are the safest of the year, for children as well as adults.

MARKLE FOUNDATION ANNOUNCES FIVE-YEAR GRANTS FOR TWENTY-ONE SCIENTISTS

Twenty-one doctors, all faculty members of medical schools in the United States and Canada, have been appointed as the sixth group of Scholars in Medical Science by the John and Mary R. Markle Foundation. Toward the support of these doctors and their research, the Foundation has appropriated $630,000, to be granted at the rate of $6,000 annually for five years to the twenty-one medical schools where they will teach and carry on medical research. In announcing these new appointments John M. Russell, Executive Director of the fund, said that a total of over $3,200,000 has been appropriated toward the support of 111 doctors in 55 medical schools since the Scholar program began in 1948.

The purpose of the program is to help relieve the shortage of medical school teachers and investigators by offering both academic security and financial aid to faculty members at the start of their careers in academic medicine. The present group of twenty-one Scholars was selected from fifty-four candidates nominated for the grants by deans of medical schools. Four regional committees of laymen aided in the selection.

The three Canadian scholars are:

1. University of Alberta Faculty of Medicine for: Robert Stewart Fraser, M.D., instructor in medicine, beginning July 1; currently, research fellow in cardiology, Medical School, University of Minnesota. (B.Sc., M.D., M.Sc., University of Alberta.) Interest: Internal medicine: cardiology.

2. University of Toronto Faculty of Medicine for: John Coleman Laidlaw, M.D., Ph.D., clinical teacher, beginning July 1, 1954; currently, research fellow in medicine, Harvard Medical School and assistant in medicine, Peter Bent Brigham Hospital, Boston. (B.A., M.D., M.A., University of Toronto; Ph.D., University of London.) Interest: Internal medicine and biochemistry.

3. McGill University Faculty of Medicine for: James R. McCorriston, M.D., demonstrator in surgery. (B.A., University of Saskatchewan; M.D., C.M., Queen's University Faculty of Medicine; M.Sc., McGill University Surgical Diploma Course.) Interest: Surgery: preoperative and postoperative care.

UNIVERSITY OF MICHIGAN SIXTH ANNUAL CONFERENCE ON AGING

"Careers for Maturing Workers" is the theme of the University of Michigan Sixth Annual Conference on Aging to be held in Ann Arbor, July 8 to 10, 1953. The problems associated with earning in the later years and with methods for creating new opportunities for remunerative activity by aging people are particularly pressing at this time because of the steadily increasing number of older workers cut off from earning opportunities.

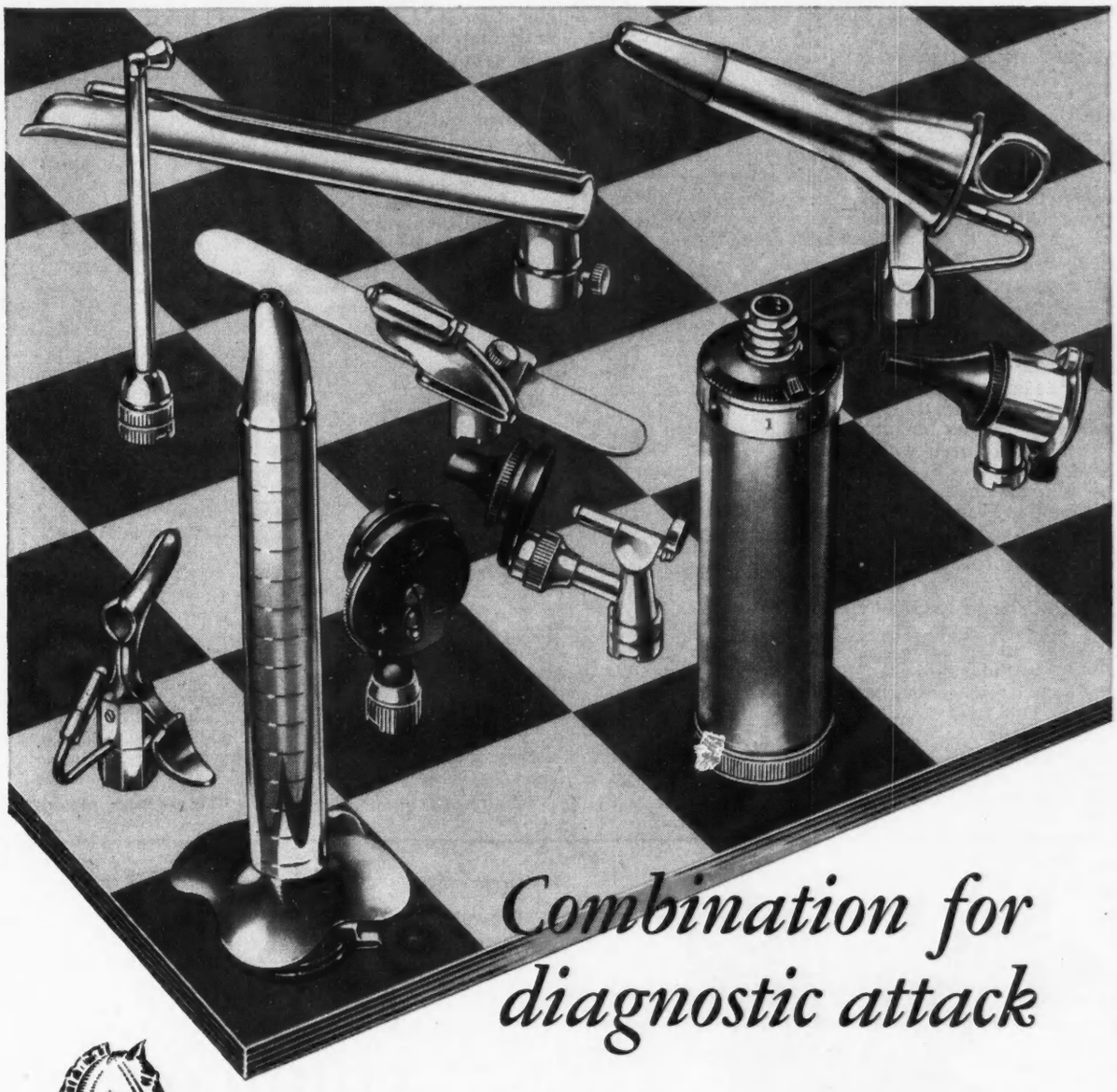
Discussion sections led by nationally known experts will consider: economic, social and personal values of continuing employment; the maturing worker from the standpoint of health; counseling, guidance, placement and training of the maturing worker; continuing employment in business and industry; needed action by business, industry, unions, communities and government agencies; and others.

Conference membership is open to anyone, but will be of special interest to industrial and geriatric physicians. Exhibits and demonstrations of marketable skills will be a feature of the Conference. For further information about the program write to: Wilma Donahue, Chairman, Division of Gerontology, University of Michigan, 1510 Rackham Building, Ann Arbor, Michigan.

INTERAMERICAN FOUNDATION FOR POSTGRADUATE MEDICAL EDUCATION

The Interamerican Foundation for Postgraduate Medical Education has been organized for the purpose of encouraging exchanges of educators, postgraduate students and research workers in the field of medicine and

(Continued on page 62 of the advertising section)



Combination for diagnostic attack

A single chess piece can be a beautiful thing, but its ultimate efficiency comes only when it is arrayed with others in a combination. Just so with diagnostic instruments. Maximum accuracy, thoroughness and speed of diagnosis are attained with instruments, each having its own function, but designed to work together as a team.

All the Welch Allyn illuminated instruments shown above, for example, are superbly designed for their particular job, yet all are instantly interchangeable on a single battery handle. This feature alone can save many minutes of the physician's time each day and reduces instrument investment by obviating the need to purchase a different handle for each instrument.

Your surgical supply dealer will be glad to give you a complete Welch Allyn catalog or to demonstrate any Welch Allyn diagnostic instrument which interests you. Welch Allyn, Inc., Skaneateles Falls, N. Y.

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NEWS AND NOTES

(Continued from page 60 of the advertising section)

allied sciences in Latin and North American countries. Substantial financial support has been obtained and more is now being solicited from commercial firms in North America interested in the furtherance of friendly relations between the Americas. In the past a number of such companies have independently supported Fellowship programs for Latin Americans who sought post-graduate training in the United States.

The new Foundation is designed to co-ordinate and extend these opportunities through a central agency which will in turn co-ordinate its program with that of other groups (private Foundations and governmental agencies) with parallel or overlapping interests in this field. Committees of medical educators in each Latin American country will be asked to assume responsibility for nominating candidates for Fellowships. The proposed program also provides for interchanges of a limited number of visiting lectures, with expenses defrayed through the Foundation. The Executive Director of the Foundation is Alberto Chattas, M.D., of Cordoba, Argentina, with present headquarters at 112 East Chestnut Street, Chicago 11, Illinois.

TUBERCULOSIS AMONG ALBERTA INDIANS

Deaths from tuberculosis among the Indians of Alberta are now about one-third as numerous as they were five years ago. The total in 1952 was 34, compared with about 90 in 1947.

An especially encouraging feature of the 1952 record is that no tuberculosis deaths occurred in three bands—the Blackfoot, Peigan and Sarcee. These bands have co-operated fully in the chest x-ray and treatment programs undertaken by Indian Health Services, and the result of their interest and co-operation is plainly evident in their health statistics. Twenty-four deaths from tuberculosis were recorded from patients in northern Alberta where less than half of Alberta's Indian population lives.

According to Dr. W. L. Falconer, medical superintendent of the Charles Camsell Indian Hospital, Edmonton, the Indian bands with the higher death rates are among the groups which resist having chest x-rays and taking treatment. Twelve of the deaths were caused by tuberculosis meningitis in children. With treatment, cures can be effected in about half the cases of tuberculosis meningitis, unfortunately, however, more than half the fatal meningitis cases did not come to hospital at all. During the past year 381 Alberta Indians received treatment for tuberculosis at the Charles Camsell Indian Hospital, the federal health department's main treatment centre for Indians living in Alberta and the Mackenzie district.

APPROVE CIVIL DEFENCE RESCUE TRAINING SCHOOL FOR ALBERTA

Construction of a civil defence rescue training school in Alberta has been made possible by co-operation of federal and provincial authorities and of the City of Edmonton, Hon. C. E. Gerhart, provincial minister of municipal affairs, has signed an agreement covering cost of the establishment which is being erected on subdivided city lots donated by Edmonton. Total cost of the project is $42,400, of which the federal government will contribute $20,000 from funds provided to assist the provinces in civil defence measures. The Edmonton property contributed is valued at $2,400. The school will be used jointly by the province and the city of Edmonton during the training of civil defence rescue instructors and rescue personnel.

(Continued on page 64 of the advertising section)

NEWS AND NOTES

(Continued from page 62 of the advertising section)

MEDICAL TEAM CO-SPONSORED BY WORLD HEALTH ORGANIZATION COMPLETES WORK IN MADRAS, BEGINS MISSION IN BOMBAY

After completing a month of work in Madras, a 14-member team of medical scientists sent to India by the World Health Organization and the Unitarian Service Committee has arrived in Bombay for four weeks of lectures and conferences, WHO's Regional Office in New Delhi has announced.

The team, which includes two Nobel Prize winners, is visiting India to exchange latest medical knowledge with Indian physicians, medical educators and public health specialists.

Its activities in Bombay will include lectures to medical audiences on subjects related to cancer, septic wounds, influenza, anæsthesia, pædiatrics, pulmonary tuberculosis and regulation of blood pressure. New medical and surgical films are being shown, and the scientists are conferring with medical personnel at hospitals and other health institutions.

The team is stressing the need for public health, preventive medicine, and the modern approach to medical education. Its discussions deal with subjects such as "The Rôle of the Hospital in the Practice of Preventive Medicine," "The Medical School and the Community," and "Solving a Community Health Problem". A field study on "Organized Home Treatment for Tuberculosis" will be made.

———

The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly in Otolaryngology, divided into two sections:

A. Basic Section, September 21 through 26, 1953, devoted to surgical anatomy and cadaver dissection of the head and neck, and histopathology of the ear, nose and throat, under the direction of Dr. M. F. Snitman,

B. Clinical Section, September 28 through October 3, 1953, consisting of lectures and panel discussions, with group participation of otolaryngological problems and current trends in medical and surgical management.

Registration will be limited. Application for attendance at one or both sections will be optional. For information write to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

———

STUDY OF MINIMAL TUBERCULOSIS

The potential seriousness of minimal tuberculosis is emphasized by Dr. Roger S. Mitchell of the Trudeau Sanatorium, Trudeau, N.Y., in reporting on a retrospective study of late results of modified bed rest in this type of tuberculosis in the April issue (Vol. 67, No. 4) of *The American Review of Tuberculosis*. The study, aided by a grant from the National Tuberculosis Association, covers 589 patients with uncomplicated minimal pulmonary tuberculosis treated with modified bed rest alone at Trudeau from 1927 through 1946.

Minimal tuberculosis, Dr. Mitchell found, "is ultimately capable of leading to advanced disease in one of four, prolonged chronic illness in one of ten, and death in one of twenty." While minimal tuberculosis under a modified bed rest regimen is affected principally by the amount and duration of disease, Dr. Mitchell says that the predominant feature of minimal tuberculosis is its unpredictable behaviour.

"The presence or absence of 'host resistance' is the reason usually given for this fact," he states. " 'Host resistance' may come and go. In most instances, however, it is quite evident that the presence of this resistance can be determined only by *post hoc propter hoc* reasoning. Is the ability to handle tuberculosis to be found in the psyche? Will further studies of corticotropin and allied substances put psychologic factors in the patient's ability to heal tuberculosis on a scientific basis? In any event, it may be concluded that, in addition to being unpredictable, minimal tuberculosis has also been essentially unsatisfactory in its response to modified bed rest treatment."